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# FURTHER STUDIES ON THE TREATMENT OF PEPTIC ULCER WITH ALUMINUM HYDROXIDE GEL*

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IT HAS become generally accepted that the neutralizing of gastric acidity is a helpful procedure in the treatment of peptic ulcer. For the average patient, the use of calcium carbonate and sodium bicarbonate, as originally advocated by Sippy, is very satisfactory and has the advantage of being quite inexpensive; but these powders are contraindicated for some patients because of the large intake of sodium. Patients with a lowered renal function may have difficulty in excreting the absorbed alkalies. Those with a tendency to form renal calculi may be made worse, and those who have a hypersecretion may require such large doses that an alkalosis develops before satisfactory control of the gastric acidity is obtained.

When aluminum hydroxide gel became available, it seemed as though this might be a useful addition to our treatment, because it was only slightly absorbed and had a fair neutralizing capacity, and because its astringent action might tend to decrease the secretion of acidity. Starting in the fall of 1937, we began to use aluminum hydroxide gel on a group of patients who were difficult to handle by means of the usual alkalies. By October, 1938, we[1] were able to show that the drug was successful in causing complete neutralization of the gastric juice, and in addition decreased the secretion of hydrochloric acid if given in sufficient amounts. In March, 1939, we[2] reported the results from treating 28 patients and were able to show that the most severe type of case can be brought under control. Having demonstrated that this drug will temporarily control severe cases, our next problem was to discover whether these patients could be satisfactorily continued on a medical treatment or would ultimately come to surgery. If the latter is true, it will save the patient time and trouble to have the operation performed as soon as possible. This paper deals with the later course of the 28 patients on whom we have previously reported.

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## PRESENTATION OF DATA

As previously stated,[1] 16 of these cases represented the severest type of the disease, while 12 were classified as moderately severe. We purposely eliminated the mild cases because they will respond satisfactorily to any good medical or surgical regime. Nine of the patients were observed for only a month. Two of these were started on the treatment in anticipation of surgery, so that we might determine what effect aluminum hydroxide had on the mucous membrane. Two patients who followed the treatment for a month in the hospital failed to return for further observation in the Outdoor Department. Five returned once, were feeling perfectly well at the time and never came back. Four patients were followed for three to five months. They were all feeling well at the time they were last seen, and the gastric analyses showed a lower titration of hydrochloric acid than before starting the treatment.

This leaves 14 patients who were followed for eleven to eighteen months, with an average of fifteen months. Eight of these have had no symptoms and can be considered well. Two are classified as in satisfactory condition; they have had a return of mild symptoms on one or two occasions. The condition of 4 is unsatisfactory; 1 is dead, 1 has had a massive hemorrhage, and 2 have had a return of active symptoms.

Those patients who fall into the "well" group took aluminum hydroxide every hour throughout the day for four or five months. After this, they were given a regime of milk once between meals and a dose of aluminum hydroxide five or six times a day, so that all have taken a maximum of six doses a day for a minimum of three months. Five cases in this group were originally classified as of the severest type, and 3 were called moderately severe.

Of the 2 patients who showed satisfactory results, the first had had a subtotal gastrectomy performed for a carcinoma in the pyloric region. He later developed a marginal ulcer and complained of pain, particularly at night. Aluminum hydroxide was allowed to drip into the stomach continuously for one week, following which he took the medicine at hourly intervals throughout the day. Under this regime he was completely free of all symptoms for one month. Since then he has had occasional discomfort at night, which is promptly relieved with a dose of aluminum hydroxide. At no time have his symptoms been so severe as they were before he started on aluminum hydroxide. The second patient was a woman who received the drug hourly for four months. For the next eleven months she took it six times a day, with complete relief until her child became seriously ill with pneumonia. During this period of worry and marked fatigue she stopped taking the medicine, with the result that she suffered a return of some mild symptoms. These were promptly relieved by the resumption of hourly doses of the drug. This case was originally classified as the severest type.

Of the 4 unsatisfactory cases, the first was originally classified as severe. The patient always had difficulty in taking the medicine and was unable to keep the tube down during the day, so that she was started with hourly feedings and the drip method was utilized only during the night. After a week and a half of this therapy her acid dropped from 138 to 95, but she remained on hourly feedings for only three weeks, at the end of which time she went back to the usual calcium and soda powders. A year later she re-entered the hospital with marked symptoms. A gastric analysis at this time gave a free acidity as high as 170. The patient was unwilling to try aluminum hydroxide therapy again. As we were not accomplishing anything with the ordinary alkaline powders, she was transferred to the Surgical Service. A subtotal gastrectomy was performed and she died four days postoperatively.

The second unsatisfactory case was that of a man thirty-five years old, and was classified as severe. A gastrectomy had been performed eleven months previously. A gastrojejunal ulcer developed which was not satisfactorily controlled by the ordinary alkaline therapy, and the patient was placed on aluminum hydroxide. He followed the treatment intensively for seven months, during which time he felt perfectly well. He was then transferred to six doses during the day, but at the end of seven months had a massive hemorrhage. This particular attack of bleeding followed an orgy of beer and scallops. He was then advised to take hourly doses of aluminum hydroxide, but there was some doubt in our minds as to how thoroughly he followed any regime. At any rate, he admitted not having taken the medicine as instructed, and five months later he had another hemorrhage.

The third case was originally classified as moderately severe, and the patient was completely relieved while taking the drug hourly, which he did for the first five months. He was then transferred to six doses a day and for a period of time continued to be free of symptoms. He then developed a marked mental depression which was associated with a return of the gastric symptoms. In spite of this fact, the free acidity remained considerably lower than it was before the treatment was started. Before treatment was instituted it was 100, and seventeen months later only 52.

The fourth case was originally classified as of the severe type. The patient followed intensive treatment for two months, at the end of which time he felt well. Then for fourteen months he took the treatment in a very haphazard way, paying little attention to any advice which was given him. He was very neurotic and it was difficult to evaluate his complaints. His first gastric analysis showed a free acidity of 100, and one done only six months later was 48.

Of these patients who showed unsatisfactory results, 1 never took proper treatment; 2 were well so long as they followed treatment, and trouble developed only when they did not take reasonable care of themselves; 1 developed symptoms after the onset of a marked mental depression. The gastric acidity has been checked in the 18 cases which were followed for three to eighteen months. In 14 cases it remained lower than before the treatment was started. The average before treatment was 97, but fell to 48 at the time of the last observation.

## Discussion

Interest in this group of patients centers around the 14 whom we have been able to follow for an average period of fifteen months. The others have not been observed long enough to permit us to draw any worth-while conclusions. Eight patients followed for the longer period are well; 2 are in a satisfactory condition; and 4 have had unsatisfactory results. The striking fact shown by these figures is the high percentage of well patients in a group which was chosen for the severity of the disease. It is to be expected that better results will be obtained by any form of treatment on patients who are being closely followed. The very fact that the patients are cognizant of the interest being shown in their progress causes them to follow treatment more assiduously than if they were not watched so closely. On the other hand,

it must be remembered that if we had not been interested in determining what could be accomplished by the use of aluminum hydroxide, subtotal gastrectomy would have been advised for all but possibly 3 of these patients. Past experience has taught us that the usual medical treatments accomplish so little with this type of case that surgery will save time and trouble. In this connection, it is interesting that the only patient who completely failed to follow treatment ultimately came to a subtotal gastrectomy and suffered a postoperative death.

It must be emphasized that the use of aluminum hydroxide is only another way of neutralizing gastric acidity, and does not hold out any greater hope for an ultimate cure than do the other methods of antacid therapy. But it is our opinion that aluminum hydroxide is the most effective drug that we have found to date for controlling severe cases during a period of acute activity. It now appears that if special attention is given to these patients, many of them can be carried along satisfactorily on a medical regime, provided they are willing to take five or six doses of the gel a day. Also, it is our present opinion that these patients have done better by continuing on five or six doses of aluminum hydroxide than if they had reverted to the usual calcium and soda powders. This may be due to the aluminum's maintaining the gastric acidity at a lower level than if the patient had taken some other form of alkali. We say this because it is our impression that patients with similarly severe cases who have been treated by the usual alkalies and have been temporarily relieved of their difficulties have not continued to do so well as did this group of patients. However, this is mere speculation, because we are not acquainted with any data which tell us whether the severe type of case, during a period of acute activity, shows an excessively high secretion which will later drop after the acute activity has subsided.

The work of Brown and Dolkart[3] suggests that there is no relation between the secretion of hydrochloric acid and the relative activity of the ulcer, but their observations do not specifically cover this type of case.

In view of the observations which we have made to date, we are inclined to give the severe type of case a trial with aluminum hydroxide before advocating subtotal gastrectomy. We say this not only because of these results, but because we have never been so enthusiastic about subtotal gastrectomy as have many other clinicians. There is always great enthusiasm for some new method of therapy, which usually decreases after a sufficient time has elapsed for a proper evaluation of results. As one of us has continuously pointed out, subtotal gastrectomy interferes with the efficiency of digestion and, by and large, patients are better off with their stomachs than without them. Although our clinic has been very conservative about the use of radical surgery, we have already had enough undesirable results to make us believe that this type of therapy should be used only as a last resort.

## Summary and Conclusions

The results from the use of aluminum hydroxide therapy on 14 cases with peptic ulcer of the severe type are reported.

After these patients had been observed for an average of fifteen months, 8 were found to be well. Two others were getting along satisfactorily, although they had had a temporary return of mild symptoms, and 4 were in an unsatisfactory condition.

These results are considered very gratifying in view of the fact that all these patients were chosen for treatment because of the severity of their disease.

It is suggested that treatment with aluminum hydroxide has a definite place in the medical therapy of peptic ulcer, and that patients should be given the advantage of this therapy before subtotal gastrectomy is advocated.

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# OMENTUMECTOMY AS A SAFEGUARD AGAINST THE RECURRENCE OF OMENTAL ADHESIONS*

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PROBABLY the patients who cause more worry to the surgeon and suffer more chronic miseries than do any others are those who have omental, visceral, parietal or pelvic peritoneal adhesions which have been caused by peritoneal injuries.

It has been my custom in cases where no acute infection was present and the omentum was found adherent to the pelvic organs, to free the omentum and remove enough of it so that the remainder was too short to reach into the pelvis, and therefore could not readhere to the pelvic organs. The patients who had omental adhesions were usually those who had had one or more previous operations or suppurative conditions such as salpingo-oöphoritis or appendicitis. The omentum was removed as the first procedure in these cases before the other lesions were taken care of. In my private practice, I have continued to cut off the omentum in such cases, but as the years have gone by, the tendency has been to remove the omental apron at a higher and higher level.

The American literature reveals no one advocating the removal of the omentum, but on the contrary lays stress on the important functions that are performed by this structure; such profound silence suggests that the procedure is so radical and would be followed by such dire results that the subject should not even be discussed.

The almost universal practice seems to be to let the omentum alone except where by its adherence it is causing definite obstructive symptoms or displacement of the organs. Under these conditions, the omentum is simply freed at the point where a dysfunction is being caused, and is then allowed to drop back into the abdomen, no attempt being made to free any more of it that may be adherent elsewhere. This procedure is still carried out, although nearly all surgeons will agree that adhesions recur in spite of all attempts that have been made up to the present time to prevent them. Their hope is that the adhesions will not again cause as severe a dysfunction as that for which they are operating, or at least that the immediate cause for operation will be relieved. They know that any new loose connective-tissue adhesions are unlikely to cause immediate severe symptoms following operation. It is only when these adhesions become old and thickened and necessarily shortened that they give rise to kinking of the bowel, intestinal obstruction, displacement of organs, occlusion of tubes and so forth.

Surgeons throughout the years have realized the seriousness of adhesions following operations and have tried all sorts of bizarre means to prevent them. Jones and McClure[1] have enumerated the foreign substances which have been introduced into the abdominal cavity in order to prevent the contact and agglutination of injured surfaces until healing can take place. To this end innumerable solutions, gases, oils, waxes and membranes — such as oxygen, nitrogen, air, salt solution, dextrose, sodium citrate, olive oil, lanolin, gelatin, paraffin, gum arabic, silver foil, gutta-percha, Cargile membranes, amniotic fluid and silk — have been tried, and they have all produced the very thing that they were introduced to prevent. These surgeons either do not know or do not believe Hertzler's[2] rule: "What the peritoneum cannot absorb it walls off or dies in the attempt. Any foreign body not capable of absorption is encapsulated." They have tried all the above methods, but would not dream of touching the omentum because they have been taught certain things in regard to it which make them loath to do so any oftener than is absolutely necessary. I shall mention several of these statements that are accepted without question, and shall attempt to prove that they are not pertinent.

Jones and McClure[1] state:

> The regularity with which the omentum is found attached about diseased foci with more or less complete walling off or isolation of the process has earned for it such striking designations as "the policeman of the abdomen" and "the abdominal leukocyte," and much has been written about its almost intelligent migratory powers.

That the omentum has the intrinsic ability at a moment's notice to rush to the site of an acute infection and wall it off is quite fantastic. Florey and Carleton[3] have shown that the movement of the omentum is entirely extrinsic. The factors influencing its change in position are

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posture, gastrointestinal peristalsis and diaphragmatic excursions. If one of these factors does not happen to bring the omentum to the site of infection, or, as is often the case, the omentum is too short to reach the focus, the intestines themselves will be forced to perform the function of walling off, and usually do.

Jones and McClure claim that the removal of the omentum is very likely to be followed by acute generalized peritonitis if the patient happens at a later date to develop a suppurative focus of infection and the omentum is not present to wall it off. I believe that the omentum plays a part in walling off areas of disease and foci of infection and often prevents generalized peritonitis, but this does not necessarily mean that the visceral peritoneum could not perform that function just as satisfactorily if the omentum were not present. It has been estimated that the total peritoneal surface area is equal to the total skin area. The peritoneal covering of the omentum makes up only a small percentage of this surface. The parietal and visceral portions of the peritoneum remaining after the removal of the omental apron have the same physiologic absorptive properties as that covering the omentum, and will respond in just the same way to infection and irritation, causing serous exudation, a pouring out of leukocytes and fibrin formation; and the contiguous inflamed or irritated intestinal surfaces will adhere and wall off the infection in the same manner as does the omentum.

Jones and McClure also state that the omentum, with its characteristic cells having distinct evolutionary history and function, is to be regarded as a parenchymatous structure analogous in some respects to the thymus gland and the bone marrow; they therefore believe that if the omentum is removed certain disease-fighting enzymes or cells will be lacking to combat any future suppurative foci of infection. Modern pathologists[4] contend that the peritoneal cells covering the omentum do not have these analogous functions.

Graves[5] points out that the omentum is covered with two layers of peritoneum, one on each side, and that it is well supplied with blood and lymph vessels, which furnish a greater amount of serum and phagocytes than does the rest of the peritoneum. This is undoubtedly true, but the visceral and parietal portions of the peritoneum have enough of these properties so that they are able to glue themselves together and surround foci of infection when they are called upon to do so. This author also states that it has been shown by experiments that animals from which the omentum has been radically removed are less resistant to peritoneal infection than are normal control animals. This may be so, but medical and surgical literature does not reveal that human subjects having little or no greater omentum have an increased susceptibility to peritoneal disease, and none of the patients from whom I have removed the omentum have developed peritonitis. The two commonest sources of suppuration in the abdominal cavity are the appendix and the fallopian tubes, and these have usually been removed at the first or a later operation. If this is not the case, the appendix should be removed and all pelvic disease corrected at the time of omentumectomy. Therefore, the danger of generalized peritonitis occurring at some future date is only remote.

Graves adds that the omentum can be used to cover denuded and injured peritoneal surfaces. This is the exact process we wish to avoid, for these adhesions cause chronic invalidism, which fact has been evidenced by the number of cases that have to be operated on again and again. He also states that it takes two injured contiguous surfaces to cause adhesions between one organ and another. This being so, it is an unquestioned fact that if the adhered omentum is freed and again dropped back into the abdominal cavity it is bound to readhere, because, first, it is a relatively immobile structure and will return by force of gravity and by the factors that influence its movements to its normal position, and secondly, the injured omental surface which was freed will very likely again be in apposition to the injured peritoneal area from which it was freed; thus the adhesion will again occur, according to Graves's statement that two injured contiguous surfaces in close apposition to one another will unite. Graves apparently did not realize the importance of his statement, for he did not utilize it in order to combat adhesions. The significant words are "two" and "contiguous." This means that if two injured surfaces are separated and kept so, or if one of them is eliminated, adhesions will not recur. The rationale of removing the omentum in order to prevent adhesions is based on these two facts. If the omentum is cut off high enough so that it cannot reach the area from which it was freed, there will be no readherence; also no adhesion will occur in the pelvis, because the raw surface there will come into contact only with the normal visceral peritoneum, which is well lubricated by serous secretion; the intestinal loops, slipping by one another and moving from place to place, will not as a rule be in contact with a mildly irritated surface long enough to unite with it.

Graves also points out that the omentum slips

into hernial openings and prevents the entrance of bowel. It is also considered a storehouse for fat, and undoubtedly acts as a blanket for the intestines and prevents their sudden chilling. These functions may well have been important in the Stone Age, but they are of little significance in the modern era of superheated homes and warm clothing.

When I perform pelvic operations, I open the abdomen by a midline incision from the umbilicus to the pubis (Fig. 1). The adherent omentum is dissected free by cutting the weblike or fibrous connective tissue that attaches it to the peritoneum. This leaves simply the peritoneal surface covered with serous cells and an overlying, practically bloodless, thin veil of cobwebby or somewhat fibrous connective tissue. This tissue has a very poor blood and lymph supply, and when cut produces slight exudation of blood and lymph, which is necessary for the formation of fibrin; there is therefore little tendency to adhere or readhere. Traction is next placed on the freed omentum, and it is pulled through the incisional wound. When it is pulled upward, the transverse colon is visualized and the attachment of the omentum to its superior border is noted. A suture is placed through the border of the omentum at the hepatic flexure, and the omentum is cut through just distal to the tie. The excision is continued along the upper border of the transverse colon to the splenic flexure, after each blood vessel that is visualized has been sutured. As soon as the heavy, redundant omental apron is removed, the whole colon, relieved of this weight, has a tendency to be drawn into the upper abdomen above the umbilicus by the shortened greater omentum, which runs directly from the greater curvature of the stomach to the upper border of the transverse colon. Consequently, there is no longer a fat pad covered with serous membrane between the small intestine and the parietal peritoneum. This leaves a thin, raw area, about 0.3 cm. thick, stretching along the whole superior border of the transverse colon. The outer and inner surfaces of this incised area consist of a thin layer of epithelial cells, and in between are fat tissue and a lacework of connective tissue; there is no injured parietal peritoneal surface in the upper abdomen to which the cut edges of the incisional wound can adhere. As a matter of fact, the cut edges naturally adhere to one another, being contiguous and raw. The injured parietal peritoneum in the pelvis comes into contact only with the visceral peritoneum, and the viscera, continually changing position, do not stay in one place long enough to adhere unless the peristaltic waves have been temporarily stopped because of shock.

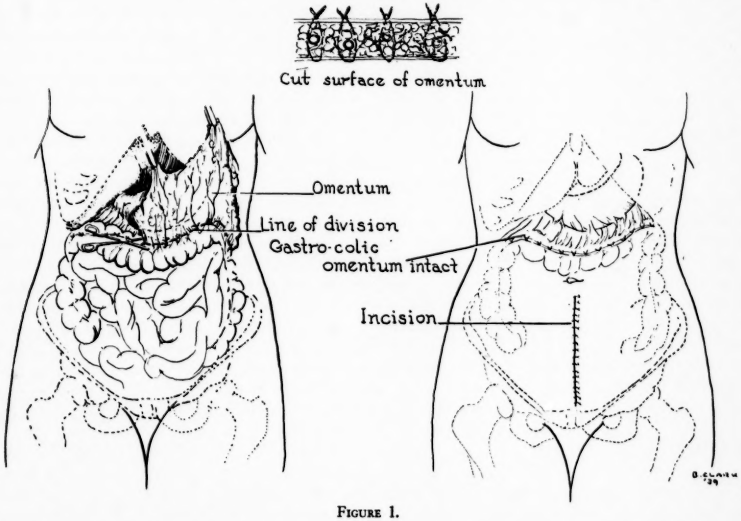


FIGURE 1.

Graves states: "The chief function of the peritoneum is to secrete enough serous fluid to lubricate its highly polished serous lining so that the

stomach and intestines are free to move around in the abdominal cavity with the greatest of ease, and so that there is no interference of the normal peristaltic movements of these organs." These movements are necessary for the proper digestion of food and the elimination of waste products, and if the omentum is drawn down into the pelvis it is bound to cause continuous pulling and kinking of the transverse colon and stomach. By compression it also interferes with the peristaltic movements of the small intestine, thus preventing the normal digestion of food and the elimination of waste products, and giving rise to all sorts of gastric symptoms, such as fullness, gaseous eructations, pain, distention and constipation.

There are other factors which must be complied with if postoperative adhesions are to be prevented:

Normally there are a few cubic centimeters of excess serous fluid in the abdominal cavity. But if a patient is debilitated or dehydrated there is less than normal serous exudation on the surface of the peritoneum, and therefore less lubrication and conversely more friction. If such a peritoneal surface is handled at operation, its delicate covering of relatively dry endothelial cells is more easily injured and it more readily adheres to another area. Therefore it is well to combat dehydration before operation by forcing fluids, and to institute routine intravenous therapy postoperatively for at least three days, until the patient is again taking plenty of fluids by mouth.

The patient should be anesthetized so that there is complete abdominal relaxation and deflation of the viscera. A basic Avertin anesthesia, supplemented by ether, or a spinal anesthesia with novocain and Pontocaine gives the relaxation that is needed if the adhesions are to be easily dissected free without causing trauma and, later, adhesions.

Finger dissection and forcible separation of adhesions by traction should never be done. Such a procedure results in more raw surfaces and thus defeats the purpose of the operation. Only the redundant part of the omentum which is to be removed should be touched by instruments or by the hand.

If the anesthesia is satisfactory, there is little or no need of walling off the viscera by the insertion of packing tapes or heavy retractors, which may in themselves cause trauma to the delicate peritoneal cells.

If clamps have to be used on the cut edges of the peritoneal incision they should be placed on the very edge, and when the peritoneum is closed these areas, traumatized by clamps, should be turned outward, leaving no injured areas along the line of suture to which the intestines may adhere.

After any complete anesthesia there is a period when the intestinal tone is entirely lost and when there are no peristaltic waves passing along the intestinal tract; thus there is very little slipping around of the loops of intestine except that caused by diaphragmatic excursions. It is during this postoperative period that the patient must be moved from side to side so as to ensure movement of the coils of intestines by change of position.

In view of the dire results that have been prophesied should the omentum be removed, the case histories of the 76 patients from whom I have removed in the last seventeen years either all or a large part of the omentum have been reviewed in order to ascertain just what good or bad results have followed. Sixty-five patients had had previous operations and most of these had subsequently been suffering from chronic gastric disturbances. Forty-one patients had had one operation, 12 two, 11 three and 1 four.

Following omentumectomy, 11 of the 76 patients had a subsequent operation. These case reports are briefly as follows:

Case 1. D. F. had an operation for peritonitis, then an appendectomy and omentumectomy for adhesions. After this she had an ectopic pregnancy and a few light adhesions were found. She has been well since then.

Case 2. B. K. had an appendectomy, then a fibroidectomy, an omentumectomy and a suspension operation. Later she had persistency of chronic metritis and underwent a hysterectomy; no adhesions were found.

Case 3. A. S. had a salpingo-oöphorectomy, then a colpotomy for pelvic abscess, then a hysterectomy, and then an operation for intestinal obstruction, with omentumectomy for adhesions. Later a laparotomy was performed for symptoms simulating intestinal obstruction, but she was evidently suffering from hysteria, since no adhesions were found, and the abdomen was sewed up without any abdominal manipulations. The patient has been well since then.

Case 4. D. C. had a salpingo-oöphorectomy and appendectomy, then a hysterectomy, and then a laparotomy and an omentumectomy for adhesions. One subsequent laparotomy was performed in order to free visceral adhesions. The patient has been well since then.

Case 5. M. B. had had an oöphorectomy and appendectomy before I performed a hysterectomy for fibroids and omentumectomy. Since then she has had two operations in a neighboring town the types of which are unknown.

Case 6. E. K. had had an appendectomy before I performed a hysterectomy for fibroids and removed the omentum. Following this she had a cholecystectomy; adhesions were found in the region of the gall bladder. Since then she has been well.

Case 7. M. F. had an appendectomy, and then a left salpingo-oöphorectomy and omentumectomy. Following

this she had a hysterectomy; very few adhesions were found. She has been well since then.

CASE 8. J. K. had had an appendectomy and suspension before I performed a left salpingo-oöphorectomy and omentumectomy. Later she had a hysterectomy for pelvic symptoms; a few adhesions were found.

CASE 9. M. R. had had an appendectomy, then a hernial repair and oöphorectomy, and then gall-bladder drainage. I did a hysterectomy for fibroids, found adhesions and removed the omentum. Subsequently the patient had a cholecystectomy; there were a few adhesions in the gall-bladder region, but none in the lower pelvis.

CASE 10. H. S. had had no previous operation until I performed a suspension and removed part of the omentum. Later she had a cholecystectomy for cholelithiasis; no adhesions were noted.

CASE 11. G. M. had had an appendectomy before I performed a left salpingo-oöphorectomy, suspension and myomectomy and removed most of the omentum. She later had an operation by another surgeon, but no report is available.

Except for Cases 5 and 11, concerning which information is lacking, it can be said that none of the 76 patients have had to undergo a subsequent laparotomy because of any complication arising from the removal of the omentum. All patients are alive except 4 who had cancer at the time of operation and had their omentums removed on account of metastases. No intestinal obstruction has occurred. No acute infection has arisen. Apparently the adhesions present at the time of operation have not re-formed to a sufficient extent to warrant further operation, with one exception (Case 4).

It would be incorrect to say that these patients have no gastric and abdominal complaints, but our follow-up shows that they are all enjoying fairly good health considering their previous chronic invalidism. Forty-four patients have reported complete relief from their gastric and abdominal symptoms, 18 partial relief and 3 no relief. Four died from carcinoma; 7 have moved away and reports have not been obtainable.

## SUMMARY

Omental adhesions cause distressing gastrointestinal symptoms and chronic invalidism.

Up to the present time no satisfactory method has been proposed that would prevent the readherence of the omentum.

Removal of the omentum is suggested as a fairly satisfactory method for treatment of omental adhesions after they have once occurred.

Case histories are cited to prove that no serious complications have followed omentumectomy.

Omentumectomy was only one portion of the operative procedure, and therefore all the good results following these operations cannot be attributed to the removal of the omentum per se, but at least it enhanced rather than lessened the patients' chances of future good health.

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## DISCUSSION

DR. GEORGE M. WATERMAN, Providence, Rhode Island: I have never encountered the total removal of the greater omentum. Dr. Larkin has given a good deal of thought to this subject, and has covered the ground and presented his argument very well. This procedure seems rather radical; however, I have had no experience with it, and Dr. Larkin has. His results, he thinks, are very good. He believes that he has had no more recurrence of adhesions, or postoperative discomfort or complications, than have surgeons who do not practice this procedure. On the other hand, he has not reported any control series which would show whether or not his argument is sound.

Personally, I shall continue to believe in the value of the great omentum, and shall be loath to remove a structure which I have always thought was a protection. This is a matter that deserves our consideration, and one concerning which we may possibly change our opinions.

DR. JOHN ROCK, Boston: I have had, unfortunately or not, no experience in the removal of the great omentum. I have, to be sure, at odd times removed a fair-sized portion of the omentum when, in separating adhesions from pelvic tumors, I had mutilated a large part of it or interfered with its blood supply. I have also removed part of it without dissecting it free from, perhaps, a fibroid, to the circulation of which it was possibly contributing a major part. More often I have used it to cover injured areas after dissection for pelvic tumor.

I think we can all agree with Dr. Larkin's very inclusive enumeration of the physiological properties of the omentum, and can agree with him that in order to exercise its function it does not have to be mobile. This is unlike the similar physiological functions of the peritoneum which covers the intestines. Dr. Larkin has also emphasized the figurative terms which have been applied to the omentum, "the policeman of the abdomen" and "the abdominal leukocyte." I must confess that I still believe these terms to be justifiable.

I have not had much to do with general surgical complications such as ruptured gall bladder or perforating ulcer, but if I had, I should hope that the omentum would be present, and I know that if ever my appendix should rupture, or that of any of my friends, — and I use the word in the Rooseveltian sense, — I should hope that the omentum would be at hand.

DR. JOE V. MEIGS, Boston: My only experience in this matter was with one of Dr. Larkin's own patients. In this case I found inside the abdomen a large, thick, smooth border of omentum that was high up. The patient was absolutely free of all adhesions, and had a perfectly

smooth omentum not over 8 cm. in length at any point. I can only offer this observation for what it is worth.

Dr. David Cheever, Boston: I will be frank in expressing doubts about Dr. Larkin's thesis. I have always considered the omentum a somewhat unaccountable but very useful structure, and having all my life preserved it so far as I could, I am not very much inclined to give up this practice. I call Dr. Larkin's attention to the fact that almost invariably when one operates on the abdomen of a person previously operated on, one finds the omentum adherent to the deep surface of the abdominal wound; the adhesion rarely, in my experience, causes any obstruction. I have always thought that this adhesion was a useful lesion and that it probably prevented the adherence of the intestine to the wound, which, in turn, might prevent normal peristalsis. My own belief is that unless the changes in the omentum are such as to indicate its removal, it should be preserved.

Dr. Larkin (closing): I agree that it is well not to be too hasty in taking up new procedures and discarding the old. But when the old procedure causes the chronic invalidism that so often follows omental adhesions I believe it is time to try omentumectomy, which, in my hands at least, has tended to prevent crippling adhesions.

## THE PROGNOSIS OF INFANTILE TUBERCULOSIS*

Clement A. Smith, M.D.,† and Winifred De Lacey‡

BOSTON

In a recent study[1] of 345 deaths from tuberculosis at the Infants' and Children's hospitals, the factor of age assumed outstanding importance. A high mortality was demonstrated in the first two years of life, as compared with a very low level in older childhood. Moreover, a significant difference in the pathologic lesions at different age levels was also apparent, deaths in infancy being usually the terminal event of miliary tuberculosis, while those of older children were much more commonly due to tuberculous meningitis not associated with miliary disease. In general, these findings have been observed by many other workers and were to be anticipated, but they increased our interest in the significance of the earliest years of life as the most valuable period for pediatric efforts against tuberculosis mortality. The importance of infancy, in contrast particularly with the later grade-school period, is shown by Table 1, with its 200 deaths in the first two years studied and its 6 deaths in the last two. In spite of the decrease in mortality during the period studied, the table shows how constantly the problem has been centered in infancy.

A natural outcome of the concentration of interest on infantile tuberculosis has been curiosity as to the fate of those tuberculous patients discharged from the Infants' Hospital during a period of years comparable to that covered by the mortality study. In the fifteen-year period from the beginning of 1924 to the end of 1938, 339 infants with some form of tuberculosis were seen in the hospital, and of this number 191 (56 per cent) died there. Data from their records form part of the preceding study. The present communication is based on a follow-up study of the remaining 148 infants who did not succumb to tuberculosis during their first hospital admission.

In almost all these infants there was roentgenological evidence of disease in the lungs or mediastinal lymph nodes, but patients with tuberculous peritonitis, cervical adenitis or bone lesions were included in the present study if they also showed evidence of tuberculosis in the thorax. Three tuberculin-positive infants with negative roentgenograms were also included, on the ground that their infection could hardly be entirely healed or inactive. In 32 cases no satisfactory data as to the course of the patient after discharge could be obtained; their omission brought the total number of records available for study down to 116. These infants were traced by letters and home visits to parents, by reports from other hospitals, sanatoriums and tuberculosis clinics, and by reference to our own records on those patients now attending the clinic. Since our first interest was in the survival of these children, we have not yet attempted to have them all

Table 1. *Deaths from Tuberculosis, 1923–1938.*

| Year | Age in Years | | | | | | | | | | | Totals |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0–1 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | |
| 1923 | 6 | 6 | 3 | 3 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 21 |
| 1924 | 16 | 15 | 6 | 1 | 3 | 2 | 1 | 2 | 0 | 0 | 0 | 0 | 46 |
| 1925 | 11 | 13 | 7 | 3 | 2 | 2 | 1 | 1 | 0 | 0 | 0 | 0 | 40 |
| 1926 | 12 | 12 | 5 | 3 | 4 | 2 | 0 | 1 | 1 | 0 | 0 | 0 | 40 |
| 1927 | 3 | 10 | 4 | 2 | 2 | 0 | 1 | 1 | 0 | 0 | 1 | 1 | 25 |
| 1928 | 6 | 8 | 4 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 21 |
| 1929 | 8 | 9 | 7 | 0 | 1 | 3 | 1 | 2 | 1 | 0 | 0 | 2 | 34 |
| 1930 | 4 | 10 | 3 | 4 | 1 | 3 | 1 | 1 | 0 | 0 | 0 | 0 | 27 |
| 1931 | 6 | 3 | 5 | 4 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 20 |
| 1932 | 5 | 2 | 4 | 2 | 1 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 16 |
| 1933 | 3 | 6 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 13 |
| 1934 | 3 | 8 | 2 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 15 |
| 1935 | 2 | 2 | 1 | 3 | 0 | 2 | 0 | 0 | 1 | 0 | 1 | 0 | 12 |
| 1936 | 1 | 3 | 2 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 7 |
| 1937 | 2 | 5 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 8 |
| Totals | 88 | 112 | 55 | 27 | 17 | 15 | 6 | 11 | 5 | 3 | 2 | 4 | 345 |

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brought back for re-examination and roentgenograms. Indeed, the impression resulting from this and similar work in other clinics suggests that the present roentgenological status of children who have now reached the low-mortality ages of later childhood would be mainly of academic interest.

An early impression which was confirmed by the results of this investigation was that subsequent deaths from tuberculosis in the later life of these infants occur almost always within a year's time. In fact, in only 1 of 42 infants who succumbed after leaving the hospital did death occur more than twelve months after the diagnosis was made. This patient lived only eighteen months. Because of this critical significance of the first year after diagnosis, we have included in our statistics the records of 22 infants whose present status is undiscoverable but who were traced and in apparently good health at least one year after being at the hospital. More than half these children had survived over five years before they were lost from the records. Since there seems to be so little evidence from the known fatalities that such patients went on to death from tuberculosis thereafter, it seemed reasonable to use them for drawing certain of our conclusions.

Data have been arranged in several tables, on which brief comment may be made. Table 2 presents a general summary; the average ages of the surviving and non-surviving patients are included, and show a slightly older age at diagnosis for infants whose course was favorable. Table 3 presents the length of known survival after the disease had been discovered, and brings out again the short period between diagnosis and a fatal termination, when that occurred.

The more important roentgenological findings at admission are shown in Table 4. Here the fatal cases are limited to those which did not have evidence of meningitis when the patients first came to the hospital. In some cases a patient was listed in more than one of these classifications, as when there were coexistent enlarged soft shadows and calcification at the hilus. By diffuse congestion is meant the frequently observed and poorly circumscribed patchy or mottled shadows suggesting the appearance of bronchopneumonia. In no case have such findings been included unless their persistence in repeated films indicated their specific etiology. It will be observed from Table 4 that 1 infant with what was assumed to be miliary disease survived, and that no patients with calcification when first seen later died of the disease. Moreover, the 3 infants with positive tuberculin tests but negative roentgenograms were all in the group with a favorable outcome.

No tabulation of later exacerbations of tuberculosis in the group of survivors is presented, nor have we exact enough data to enable us to

TABLE 2. *General Summary.*

| STATUS OF PATIENTS | NO. OF CASES | | | AVG. AGE ON ENTRY |
|---|---|---|---|---|
| | | | | *mo.* |
| Total tuberculous patients | | | 339 | |
| Died of tuberculosis in hospital | | 191 (56%) | | 11.6 |
| Discharged alive | | 148 (44%) | | |
| Untraced | 32 | | | |
| Traced | 116 | | | |
| Later died of tuberculosis | 42* (36%) | | | 11.3 |
| In less than 6 mo. | 36 | | | |
| In 6–12 mo. | 5 | | | |
| At 18 mo. | 1 | | | |
| Died of nontuberculous disease | 5 (4%) | | | |
| Survived | 69 (60%) | | | 13.1 |
| To the present | 47 | | | 13.2 |
| At least 1 yr. (present status unknown) | 22 | | | |

*Nineteen patients had miliary or meningeal tuberculosis on admission.

TABLE 3. *Length of Survival of Tuberculous Infants Living at Discharge.**

| STATUS OF PATIENT | SURVIVAL IN YEARS | | | | | | | | | | | | | | TOTALS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Under 1 | 1–2 | 2–3 | 3–4 | 4–5 | 5–6 | 6–7 | 7–8 | 8–9 | 9–10 | 10–11 | 11–12 | 12–13 | 13–14 | 14–15 | |
| Now living | 2 | 4 | 10 | 2 | 2 | 5 | 4 | 3 | 2 | 5 | 1 | 1 | 1 | 2 | 3 | 47 |
| Alive when last known | 0 | 3 | 2 | 1 | 1 | 5 | 2 | 3 | 1 | 1 | 3 | 0 | 0 | 0 | 0 | 22 |
| Died of tuberculosis | 41 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 42 |
| Grand total | | | | | | | | | | | | | | | | 111 |

*Omits 5 patients who later died of nontuberculous causes.

discuss the difficult question of the activity or healing of their earlier infections at the present time. It is known, however, that 6 of the survivors developed foci which persist at the present. These include 1 child who now has tuberculous cervical adenitis, 2 with cervical adenitis and skeletal tuberculosis and 2 with skeletal lesions only. The sixth and most interesting child has apparently developed a reinfection or adult-type lesion in the lungs. This patient was seen in the hospital in 1924 at the age of seven months, with

TABLE 4. *Outcome with Relation to Findings in First Roentgenogram.**

| Roentgenographical Diagnosis | No. of Patients | Deaths | | Survivals | |
|---|---|---|---|---|---|
| | | No. | Per cent | No. | Per cent |
| Miliary tuberculosis | 12 | 11 | 92 | 1 | 8 |
| Parenchymal consolidation | 34 | 11 | 32 | 23 | 68 |
| Diffuse congestion | 37 | 11 | 30 | 26 | 70 |
| Enlarged hilus | 10 | 1 | 10 | 9 | 90 |
| Calcification | 10 | 0 | 0 | 10 | 100 |
| Negative | 3 | 0 | 0 | 3 | 100 |

*Omits 15 patients with meningitis at first examination.

physical and roentgenological signs of dense consolidation throughout the left upper lobe, a result of intimate contact with a tuberculous mother. She spent the next two years at a sanatorium for tuberculous children, and was discharged at the age of three with regression of the lesion and the appearance of calcification. After two admissions to other institutions, mainly because of poor home conditions, the child was found at the age of fourteen to have developed a soft subapical shadow on the left, for which she was again hospitalized as having minimal adult-type tuberculosis. She has remained sputum-negative, but artificial pneumothorax has had to be abandoned because of old pleural adhesions.

## Comment

The fate of the tuberculin-positive infant has been the subject of a number of recent communications in the literature, a few of which may be briefly reviewed before making general comment on the present series.

Brailey[2] in 1936 published observations on 170 infants studied at the Harriet Lane Home in Baltimore. Somewhat more than half these patients were colored. The maximum period of observation was five years, and the material was tabulated with particular reference to the presence or absence of lesions demonstrable by roentgenogram at first admission, and to the presence of sputum-positive contacts. Sixty-seven patients had a parenchymal lesion at first examination, 36 had bulging tracheobronchial nodes and 67 had negative chest x-ray films; the mortality within these three groups was 40 per cent, 13 per cent and 9 per cent respectively. Approximately 70 per cent of all deaths occurred during the first year following the discovery of the infection. The total five-year mortality for the colored children was 31 per cent, as against 13 per cent for the white children.

As a part of Rosenberg and Kereszturi's[3] investigation of BCG in New York City, a group of non-vaccinated infected infants was observed for one to nine years. They report a death rate of 9 per cent for 348 infants followed. In the majority of cases in which roentgenograms were made, no evidence of tuberculosis was found, so that their group represents, even more than does Brailey's, a milder degree of infection than was present in our patients. All the 32 fatalities in their series occurred by the end of three years, and more than half of these during the first year. Tortone and his associates[4] have recently reported on the course of 209 infants reacting to tuberculin at the Lymanhurst Health Center. In a follow-up period averaging five years there were 10 deaths from the disease. Again, a large proportion of these patients were roentgenologically negative.

A very extensive study by Söderling[5] is based on 912 tuberculous infants observed at three hospitals in Stockholm during a period of twenty-five years. Of this large series, 487 died of their disease within three years after its discovery, 90 per cent of the deaths occurring within the first year. During the same period 13 patients succumbed to nontuberculous infections. Of the 412 surviving patients, 405 were followed for periods varying from five to twenty-five years, a record which arouses the envy of anyone who tries to trace the later course of even a hundred infants. It was discovered that only three of the 405 patients succumbed to tuberculosis during this period. In view of his observations, Söderling suggests that a considerable degree of immunity had been established by the infection in infancy.

Such statistics as those reviewed above demonstrate wide variations in prognosis, depending on the degree of involvement at the time the infection is discovered. The most satisfactory results are those from institutions whose work is largely case-finding in nature. In the earlier years of the present study, the majority of tuberculous infants died during their first admission of miliary or meningeal tuberculosis — conditions already present in most of them at entry. Those discharged alive usually represented cases of advanced disease, so that few were expected to survive. In more recent years it has been possible to discover and admit more infants in earlier stages of infec-

tion. A smaller proportion of such patients have died in the hospital; moreover, more of those discharged alive are surviving.

From all studies, including our own, it seems fairly certain that tuberculous processes in infancy run a comparatively acute course. Once an infected infant has survived a year — or perhaps two years — after the discovery of his disease, his prognosis becomes reasonably favorable and a relaxation of care and observation is permissible. It is obvious from all the references that infection beginning in the earlier months of infancy terminates in a higher percentage of fatalities than that which arises nearer the age of two years. It is also apparent that the outlook for the tuberculin-positive infant can be made more favorable than it once was. This is demonstrated by a division of the fifteen-year period of the present study into three periods of five years each, as shown in Table 5. It is gratifying to note that the percentage of tuberculous babies surviving for a year after discovery was more than twice as great for the last as for the first five years studied. However, even in the last period nearly half the tuberculous infants brought to the hospital died shortly following their first and only admission. Death often occurred only a few days after entrance, at which time the disease was usually present in a miliary or meningeal phase.

Thus, whatever improvement can be shown for prognosis is largely the effect of earlier diagnosis rather than of better therapy. The line of successful attack against tuberculosis as a pediatric problem is clearly indicated. The excellent results from Lymanhurst and from New York City demonstrate how much can be done by methods which seek out infected infants by case-finding methods before those patients would ordinarily be brought to a hospital for sick children. Children's hospitals and clinics, and private practitioners as well, should seek out the infant siblings of tuberculin-positive older children, instead of concentrating so much time and attention on those in safer age groups. Important alliances can be made between pediatric clinics and obstetric hospitals, so that the newborn infants of tuberculous parents can be regularly tested for infection during the dangerous years. To await the development of symptoms in infants before looking for tuberculosis is an even graver error than it is in adult patients. Too often the appearance of symptoms is the outward evidence that an irreversible pathologic process has begun. Responsibility for diagnosis before the onset of this phase rests on the physician and the clinic. The means of diagnosis are the family history, the tuberculin test and the roentgenogram. The results of such efforts should be not only an ultimate diminution in the number of tuberculous infants, but also a better prognosis for those discovered.

TABLE 5. *Comparison of Results in the Three Five-Year Periods.*

| Results | Periods of Admission | | |
|---|---|---|---|
| | 1924–1929 | 1929–1934 | 1934–1939 |
| Total tuberculous patients | 173 | 100 | 66 |
| Died in hospital | 106 (61%) | 56 (56%) | 29 (44%) |
| Living at discharge | 67 | 44 | 37 |
| Untraced | 24 | 6 | 2 |
| Traced | 43 | 38 | 35 |
| Died after discharge | 17 | 16 | 9 |
| Died of nontuberculous disease | 3 | 1 | 1 |
| Survived for over 1 yr. | 23 (54%) | 21 (55%) | 23* (66%) |
| One-year survivors | 13% | 21% | 35% |
| Total mortality due to tuberculosis | 71% | 72% | 58% |

*Actually 25 survivors, but 2 have not completed the full year.

## Summary and Conclusions

Since the great majority of deaths from tuberculosis during infancy and childhood occur in the first two years of life, the attention of pediatricians must be centered on infancy as the most important age period for further lowering the mortality from this disease. A study of the outcome of tuberculosis in 339 infants over a period of fifteen years has demonstrated a 56 per cent mortality for the group during the first admission to the hospital. Of the 116 remaining patients whose course is known since discharge to other institutions or to their homes, more than half have survived for one to fifteen years after the original diagnosis was made. In all but 1 of those who succumbed to their disease this event occurred within twelve months; 1 infant died after eighteen months. Five later developed a skeletal or glandular tuberculosis, and 1 an adult type of pulmonary tuberculosis.

A review of other reports in the literature indicates a wide variation in the prognosis for the tuberculous baby. The mortality varies directly in proportion to the degree of disease evident at discovery. In the present study almost all in-

fants had roentgenologically significant disease at that time, but efforts directed toward earlier recognition of infection in recent years have been rewarded by a significant increase in the number of survivors. Such efforts must be made by private physicians, clinics and other institutions aware of the value of diagnosis by family history, tuberculin testing and roentgenography before symptoms appear.

Attention is called to the acute nature of infantile tuberculosis, and to the attendant improvement in prognosis once the infant has survived the critical year after discovery.

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## GANGRENE OF THE SCROTUM AND REPAIR BY A SIMPLE PLASTIC OPERATION*

### Report of a Case

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IT IS not the purpose of this paper to discuss in detail the various types of gangrene of the scrotum, but simply to give a brief outline of the various methods, to report a case and its preoperative treatment, and to describe a simple plastic operation.

Gangrene of the scrotum may be classified under two headings.[1] To the first belongs secondary gangrene following infectious diseases, such as diabetes, the terminal stages of gangrene following some total pathologic change, such as abscess and cavernositis, and cases of mechanical origin due to chemicals, excessive heat or frostbites. To the second group belong the so-called "idiopathic" or spontaneous types of gangrene known as streptococcal gangrene and gangrenous erysipelas. As regards the latter group, there can now be little doubt as to the infectious nature of the condition. We know that there is a gangrene produced by gas-forming organisms, as well as the cases of streptococcal gangrene. It is my belief that the term "idiopathic" is rather loosely applied, and that such cases are for the most part examples of symbiotic gangrene.

The case here presented falls into the last-named group. The early pathologic changes are striking, with a close resemblance to erysipelas. There is sudden acute inflammation at the onset. The skin becomes reddened, tense and glossy, and there is usually no evident portal of entry for the organisms. Campbell[2] states that the mode of entry is unknown, but that it is assumed that the organisms are rubbed in by friction of the clothing. There may have been some abrasion to the skin or urethra, unknown to the patient. The surface readily pits on pressure, with the appearance of an exudate and desquamation of the dependent parts. Following this, the gangrene may spread rapidly in all directions. A line of demarcation may soon appear, or the gangrene may spread to the groin or even upward to the umbilicus or axilla. Extension usually follows the fascial planes of Scarpa and Colles. If untreated, the slough soon separates, usually in two or three days, and comes away as a foul-smelling mass, leaving the testicles and penis relatively free of involvement. However, as was noted in the present case, there is frequently exposure of vessels, with the subsequent danger of hemorrhage. With proper care, improvement may immediately be noted, with rapid granulation and in time even complete regeneration of the scrotum.

The general methods of treating cases of scrotal gangrene have for the most part been fairly satisfactory. Wide excision of the involved skin and subcutaneous tissues, with continuous postoperative hot dressings of a 1:5000 solution of potassium permanganate, has seemed to work very well in many cases.

Just how important a part antistreptococcus serum may play in the treatment of some of these gangrenes, I am as yet unable to state; and the same holds true for the use of sulfanilamide. I think that at the present time by far the most effective drug to be used in cleaning up the infection is zinc peroxide.

The active treatment with zinc peroxide should not be started until the determination of the bacteriological flora of the wound has been reported.

*Presented at a meeting of the New England Branch of the American Urological Association, Boston, April 20, 1939.

†Surgeon-in-chief for urology, Boston City Hospital.

This type of treatment should be used only when the organisms which are found are known to be susceptible to peroxides; such organisms include the anaerobes, the microaerophiles and certain aerobes, including the ordinary hemolytic streptococcus. It is a well-established fact that certain of the so-called anaerobes are strictly anaerobic in their cultural requirement, while others seem to be in an intermediary group, growing under a slightly reduced oxygen tension and later adapting themselves to aerobic cultivation. These bacteria have been referred to as microaerophilic.[3] Frequently it is possible to make a clinical diagnosis of anaerobic infections. However, anaerobic methods of bacterial cultivation are obviously necessary for accurate determination of the nature of the infection.

When the presence of these organisms has become established, there must be a preliminary surgical procedure which will render it possible to apply medication directly to the affected tissue in such fashion that the main focus will be removed, and the material then have access to the residual infection.

Zinc peroxide has been used also as a prophylactic in surgical wounds, where contamination with anaerobic and microaerophilic organisms is suspected. The prophylactic use of the drug is more logical than is its use for active treatment, as the organisms have not yet invaded the tissues. While treating active infections, zinc peroxide must be applied to every part of the wound surface, because its action does not extend far from the point of local application, as has been demonstrated in vitro.

The work of Meleney and Johnson[4] indicates that the liberation of oxygen and production of hydrogen peroxide depend on the presence of water. They therefore suggest that the zinc peroxide be applied suspended in sterile distilled water, with the dressing sealed in order to prevent evaporation.

Zinc peroxide is finely divided white powder, having the consistence of chalk or talcum. It must be heated at 130 to 140°C. from one to four hours in order to sterilize it and to mobilize the oxygen. For use it is mixed with roughly ten parts by weight of distilled water[6]; this is accomplished by means of a sterile glass syringe, the result being an even, creamy suspension. More water may have to be added during the dressing.

The mixture is injected from the syringe so that it comes in contact with every point of the infected surface, especially beneath the undermined skin and down into the sinuses, using a small catheter when necessary. Where the zinc peroxide does not reach the organisms, they will continue to grow. Strips of gauze or silk are dipped in the suspension and introduced under the skin edges until the wound is completely covered, including the edges of the normal surrounding tissue. The dressing is then sealed with a double layer of vaselinized gauze on zinc-oxide-impregnated gauze. The dressing should be changed daily and the old zinc peroxide washed away with sterile water or saline solution.

In the case about to be reported, there was a very marked improvement at the end of three days. The exudate decreased, and granulations appeared and became very firm. At the same time extension of the infection stopped abruptly, and the undermined edges were sealed off almost overnight. There were, however, a few areas which persisted for several days, appearing as whitish-gray patches associated with discharge but these responded well to treatment in a very short time (Fig. 1).

FIGURE 1. *Preoperative Lesion.*

*This photograph was taken 24 hours prior to operation on the day the zinc peroxide dressings were discontinued.*

## CASE REPORT

J. D., a 48-year-old, Italian-born cook, was admitted to the Boston City Hospital on April 13, 1938, complaining of pain, redness and swelling of the scrotum of 3 days' duration. He stated that he had noticed a swollen, nontender "pimple" on his scrotum intermittently for several years. However, this had given him no trouble until 3 days prior to entry, when, while working over a hot stove, he noticed a burning sensation in the scrotum and began to feel weak and sick. That evening he discovered that the scrotum was red and tender. He was seen by his physician, who advised the application of icebags, and, as the condition became more severe, referred the patient to the hospital. The past history was essentially negative. The patient had had one attack of hematuria 8 years previously. He had had gonorrhea in 1917.

Physical examination revealed a robust, healthy-appearing man with marked dental sepsis. The general examination

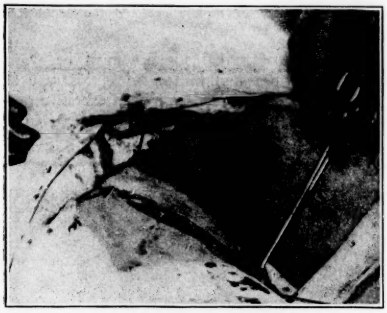

FIGURE 2. *Technic of Operation.*

*The arrows point to the pedicle of the flap on the right thigh and the line of incision for the flap.*

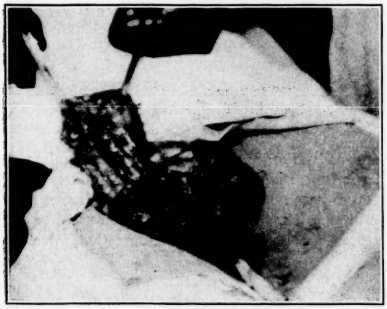

FIGURE 3. *Technic of Operation.*

*The flap on the right thigh has been dissected free to the pedicle.*

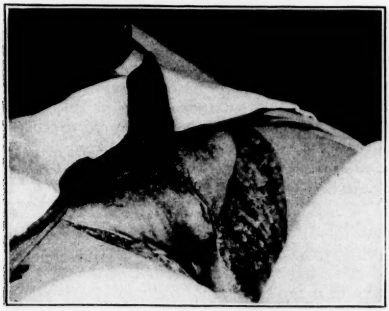

FIGURE 4. *Technic of Operation.*

*The flaps from the right and left thighs have been reflected over the testicles and stitched together in the midline.*

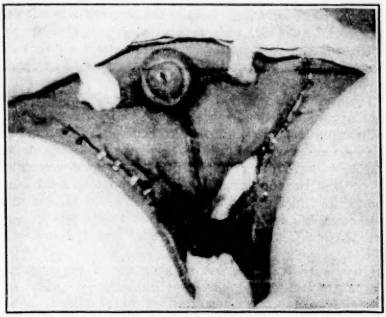

FIGURE 5. *Technic of Operation.*

*The skin defects have been obliterated, gauze drains inserted, and the final stitches and skin clips placed.*

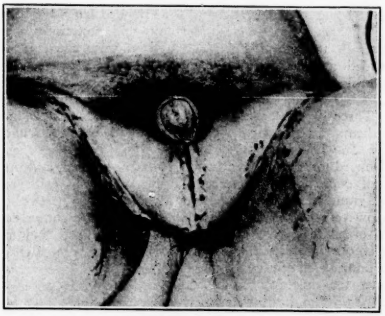

FIGURE 6. *Result on the Day the Stitches and Clips Were Removed.*

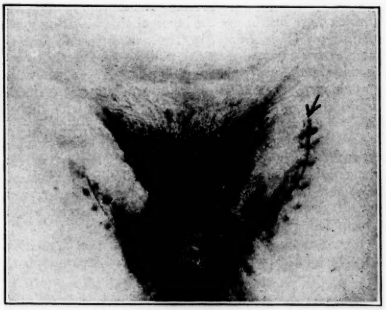

FIGURE 7. *Result on the Day the Stitches and Clips Were Removed.*

*This view shows the pedicles of the flaps before they were severed.*

was otherwise negative. Locally the scrotum was edematous and hyperemic, with a denuded area approximately 6 cm. in diameter, around which there was an area of necrotic tissue with surrounding inflammation. The entire involved area was moderately tender.

Laboratory examination showed an alkaline urine containing no sugar or albumin. The sediment was free of cellular elements at all times. The hemoglobin was 65 per cent, the red-cell count 5,370,000 and the white-cell count 14,000, with 75 per cent polymorphonuclear neutrophils, 17 per cent lymphocytes, 3 per cent eosinophils, 3 per cent monocytes and 2 per cent basophils. A blood Hinton test was negative. The blood nonprotein nitrogen was 35 mg. per 100 cc. The fasting blood sugar was 132 mg. per 100 cc.

On admission a diagnosis of gangrene of the scrotum was made and the scrotum was immediately incised in several places. Chlorinate dressings were applied; however, the necrotic tissue continued to increase. There was no evidence of urinary extravasation. On the 4th hospital day the entire scrotum was involved and the process had started to spread to the thighs and abdominal wall. A culture was taken at this time. A complete debridement was then done, removing the gangrenous tissue completely to the line of demarcation, which extended from the base of the penis anteriorly to the anus and posteriorly and laterally to both thighs. The testicles were entirely denuded and seemed to be involved by the gangrenous process. However, they were left intact. A zinc peroxide dressing was applied, following the prescribed method.

Three days later the culture was reported positive for a hemolytic streptococcus with anaerobic properties and for *Staphylococcus aureus*. The white-cell count had dropped to 9400. The area was remarkably clean, and the foul odor had disappeared. There was early evidence of granulation and no further spread of the gangrene. However, daily zinc peroxide applications were continued in order to ensure destruction of all organisms and to promote granulation. The underlying margins gradually became sealed off, with signs of epithelization. Two weeks after the debridement the area was completely covered by fresh and healthy granulation tissue and was free of organisms by culture.

Twenty-eight days after debridement there was but little evidence that the scrotum would regenerate. A plastic operation was done, at which time two large pedicle flaps were freed from the inner surface of the thighs, the bases of which were just below the inguinal line, ensuring good blood supply (Figs. 2 and 3). These flaps were so formed that they were turned over the testicles, completely covering them and forming a sac (Fig. 4). The edges were approximated by the use of silkworm-gut stay sutures, reinforced by skin clips. The sac was drained with rubber tissue. The denuded area left by the formation of the flaps was completely closed at this time, this being possible because of the loose skin associated with loss of weight by the patient (Fig. 5). Two gauze drains were placed under each pedicle and a snug, dry sterile dressing was applied. A Foley self-retaining catheter was inserted to aid in maintaining cleanliness at the operative site.

There was no postoperative sepsis, and the circulation was very good. The pedicles were severed separately and the newly formed sac rapidly assumed the appearance of a normal scrotum (Fig. 8).

Some type of scrotal support must be worn for at least three months following this type of plastic operation in order to prevent excessive sagging.

Many of these cases heal without a plastic operation, but there are a certain number of cases, such as the one reported, in which, unless a plastic operation is performed, the testes and scrotum become so bound down by scar tissue that the urethra is involved, thus causing a stricture that later on in life may require operative interference.

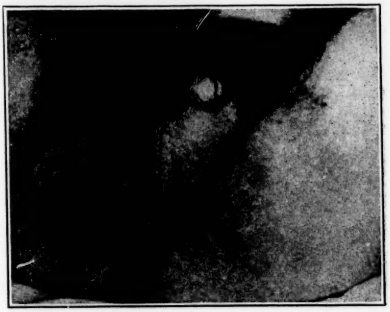

FIGURE 8. *Final Result.*

## SUMMARY

A case of extensive gangrene of the scrotum is reported, the treatment by the excision of necrotic areas and the use of zinc peroxide in cleaning up the infection is discussed.

A simple plastic operation for reconstructing the scrotum is described, which should be used only in those cases of extensive gangrene that may lead to stricture formation.

270 Commonwealth Avenue.

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# URETHRITIS FOLLICULARIS*

ISADORE J. ZIMMERMAN, M.D.†

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AVAILABLE published works, including textbooks, are rather vague in their description and evaluation of the nodular or polypoid protuberances frequently encountered at the proximal portion of the female urethra and bladder neck. These nodules are visualized with the foroblique telescope and the McCarthy cystourethroscope by withdrawing the instrument into the urethra with the irrigating fluid actively flowing. Some are club-shaped, with a small pedicle; others are blunt, with a broad base. While they appear to be more commonly situated near the bladder neck, they may extend for a variable distance into the urethra. The nodules are covered by normal-appearing mucosa. Careful observation usually reveals one or more blood vessels entering them at the base. These projections have been frequently referred to as inflammatory tags or polyps of the female urethra. Because of their obviously benign character, they have seldom been subjected to biopsy.

I have removed a few of these nodules from 3 women for microscopic study. The patients were in their third, fourth and fifth decades respectively. All 3 showed infection of the uterine cervix and pus and bacilli in the urine obtained by catheterization. Two had cystoceles with incontinence. One had a proved papilloma of the bladder near the right ureteral orifice, and a right pyelonephritis presumably associated with the mild obstruction occasioned by the papilloma. No patient had at any time had hematuria, although it was present in several of the reported cases of follicular lesions elsewhere in the urinary tract. The biopsy specimen in each case revealed a solitary lymph follicle with densely packed lymphocytes at the periphery and a typical germinal center (Fig. 1).

This lesion is not confined to the female urethra. Figure 2 shows the typical lymph follicle from a presumed polyp removed from the prostatic urethra near the internal orifice of a sixty-two-year-old man with a profound bacillary infection secondary to an infected hyperplastic prostate.

In one of the sections taken from the female urethra, lymph follicles and glandular structures were found in the same polypoid mass (Fig. 3). In the gross this structure differed not at all from the others.

*Presented at a meeting of the New England Branch of the American Urological Association, Boston, April 20, 1939. From the Urological Service of the Sacred Heart Hospital, Manchester, New Hampshire.

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Since there is a paucity of literature on urethritis follicularis, it should prove profitable to review briefly some of the more recent reports on follicular lesions elsewhere in the urinary tract. Kretschmer[1] in 1913 reviewed the literature on pyelitis follicularis and found only 7 cases. Of these the bladder was involved in only 2 cases, 1 of these being Kretschmer's. Hundley and Carson[2] in 1929 added 3 more cases of pyelitis follicularis to the literature. Hinman and Cordonnier[3] studied 7 cases of cystitis follicularis and biopsied 5. Six patients were women and 1 a man. The ages ranged from eight to sixty-nine, although most of the patients were over forty. Colon bacilli were present in the bladder urine at intervals in all the cases. Colon bacilli were also reported in Kretschmer's case and in that of Von Frisch. Busch[4] in 1905, by making serial sections of the urethra of cadavers, discovered lymph follicles in the fossa navicularis in 6 adult men ranging in age from twenty-five to sixty-seven years. He found none in the fossa navicularis of 2 newborn infants.

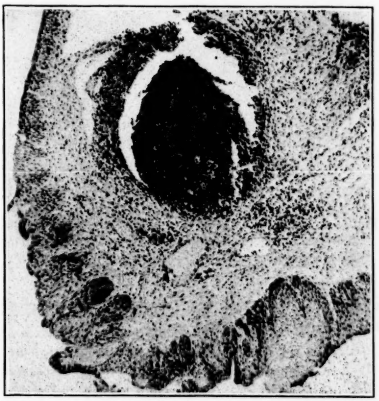

FIGURE 1. *Solitary Lymph Follicle in a Polypoid Mass Removed from the Proximal Portion of the Urethra in a Woman.*

Pelouze[5] reported 75 cases of what he designated as lymphocystic urethral lesions at and distal to the margin of the vesical outlet and attributed them to active tuberculosis. He later modified his

view when he was able to find no demonstrable tuberculosis in some of the cases. In such patients, however, he found a concomitant prostatitis which could not be cured so long as the lesions were present. The lymphocystic bodies of Pelouze have been classified by Hinman midway between cysts and polyps. Because cystic degeneration of lymph follicles elsewhere in the body rarely, if

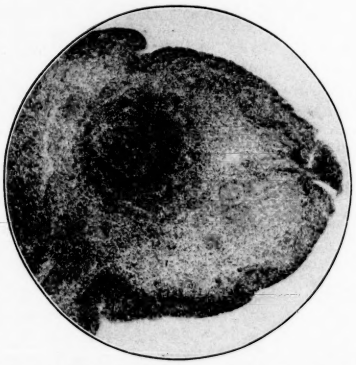

FIGURE 2. *Lymph Follicle in a Polypoid Mass Removed from the Prostatic Urethra in a Patient with a Profound Bacillary Infection.*

ever, occurs, it does not seem proper to include them in the follicularis group.

Stirling[6] recently added 4 cases of cystitis follicularis to the literature. One case was associated with cystitis cystica, cystitis glandularis and pyeloureteritis follicularis, and 1 exhibited cystitis follicularis and cystitis glandularis in the same specimen. In each case the urine was infected with colon bacilli.

True solitary lymph follicles have been found, then, in the renal pelvis, the ureter, the urinary bladder, the posterior urethra and the fossa navicularis.

Urethritis follicularis must be differentiated from the cystic, glandular and granular groups (with which it may, however, be associated), from true polyps, small fibromas or myomas which are probably not very common and from bullous edema. Because clinical differentiation may at times be difficult and because the lesion may have special significance in relation to associated pathologic lesions, exact diagnosis and therefore a rational therapeutic approach demand more frequent biopsies. Chronic infection, although not mentioned in every reported case presenting clinical lymph follicles, appears to be an important factor.

Whether submucosal lymph follicles are normally present in the urinary tract has been a subject of considerable interest. Time does not permit a detailed discussion of this problem, which is ably presented by Busch, Kretschmer, Hinman and others. Lymph follicles have as yet not been reported in the urinary mucous membranes of infants. The manifestation of enlarged follicles in the presence of infection, however, would seem to point to the pre-existence of occult lymph tissue in the urinary mucous membranes, with perhaps individual variation. Inasmuch as solitary lymph follicles occur in the mucous membranes of various other organs and because of the close embryological relation between the urinary tract and the alimentary tract, the absence of nodules appears

FIGURE 3. *Glandular Structures in a Polypoid Mass Removed from the Proximal Portion of the Urethra in a Woman.*

*The same specimen also showed two lymph follicles.*

more remarkable than does their presence. Their spontaneous disappearance from the bladder with the subsiding of infection has been reported. In this respect they do not differ greatly from the other proliferative lesions of the mucous membranes of the urinary tract. This phenomenon in each case suggests the necessary presence of a preclinical structure made manifest by the presence of infection. MacCallum,[7] quoting Ribbert, points out that there are many scattered lymph follicles in the tissues which become evident only when they are caused to swell by some inflammatory reaction.

Not all patients with chronic genital or urinary

infection develop follicular manifestations. One or more additional coexisting factors must of necessity, therefore, be involved. Of these, a wide individual variation in the presence of submucosal lymphoid tissue in the urinary tract has been predicated as a most reasonable explanation. Recent experimental work with estrone in monkeys, while in no way refuting this theory, is of considerable interest.

Sandys and Zuckerman[8] demonstrated small lymph nodules deep under the urethral epithelium in male rhesus monkeys, and showed that these nodules became manifest after the monkeys had been injected with estrone. The nodules increased in extent as long as the injections were given. Further studies on the relation of these follicles to hormonic influence and their possible relation to general and local defense mechanisms against infection and toxic products would be enlightening.

From available evidence I am led to look upon these follicles as secondary rather than primary manifestations of infection. Their presence, therefore, whatever the associated symptomatology may be, should lead to a search for the primary cause, particularly in the urinary and the genital organs of both men and women. Being cognizant of the facts that lymphoid tissue is part of the body's defense mechanism against bacterial invasion and that the follicular nodules may be expected to disappear with the eradication of the primary source of infection, it would hardly seem logical to fulgurate them indiscriminately as polyps. If, however, by their location they interfere with the complete emptying of the bladder or are a source of irritation or disturbing hemorrhage, complete destruction with high-frequency current would appear to be indicated.


## Summary

Microscopical study of polypoid masses removed from the posterior urethra of 3 women and 1 man showed the presence of true solitary lymph follicles in each case and associated glandular structures in 1.

Etiologically these nodules are apparently secondary to infection elsewhere in the genitourinary tract, although certain coexisting anatomical or physiological factors probably play an associated role.

A careful study of the genital and urinary tracts in both men and women, therefore, should precede any decision with regard to the relation of these follicular lesions to urinary signs and symptoms. This point of view should lead to a rational therapeutic approach.


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### Discussion

Dr. E. Granville Crabtree, Boston: I should like to ask Dr. Zimmerman if these cases are sufficiently differentiated for them to be distinguished from round-cell infiltration.

Dr. Zimmerman (closing): The differential point between true lymph follicles and round-cell infiltration is the germinal center. In each of the two sections shown there was a light central portion and a darker peripheral area. The lighter central area is the germinal center, which is not present in round-cell infiltration.

# REPORT ON MEDICAL PROGRESS

## SYPHILIS

C. GUY LANE, M.D.,* AND G. MARSHALL CRAWFORD, M.D.†

BOSTON

SYPHILIS has been widely discussed in the past year as a result of the campaign of the United States Public Health Service, both in the medical and in the lay press, for its eradication. While only a few of these articles can be commented on, this publicity has resulted in wider discussion of the disease by the general public and greater familiarity with its manifestations and treatment. Numerous laws relating to syphilis have been enacted, some of them prematurely without due consideration of all phases of the question.

### SYPHILIS AND PUBLIC HEALTH

At least forty-four states have either laws in effect or bills before their legislatures to create new laws or revise existing statutes. In Massachusetts a prenatal blood-test law became effective on November 1, 1939, and requires every physician to perform a serological test in every pregnancy.[1] The incidence of syphilis in pregnant women in various parts of the country in large clinics has been found to be from 2 to 4 per cent.[2] The fact that the loss from syphilitic blindness in the United States has been estimated at ten million dollars yearly[3] is a potent argument for such laws.

Much has been written about these laws. Kolmer's[4] article on premarital and prenatal tests for syphilis is a particularly good summary of the reasons for and against such tests for syphilis, and sums up with clarity the problem of the legal requirement of blood tests for syphilis during pregnancy and at delivery. The principle involved in the formulation of statutes for the control of syphilis has been thoroughly considered by Stokes and Ingraham,[5] who have also discussed the matter of false positive serological tests occurring in syphilis. They emphasize that laws cannot justly put the sole or major dependence on any laboratory procedure. The proper interpretation of serological tests for syphilis is vital. A single negative report does not of itself rule out the disease, and a repeated checking of positive reports in the absence of other confirmation is essential. A series of papers by Stokes,[6] Snow,[7] Nelson[8] and Towne[9] is a most comprehensive symposium on the social and public-health programs. Many phases of control, diagnosis and legal regulation are thoroughly discussed, while educational and co-operative measures are emphasized. The follow-up of the syphilitic patient is an essential feature of any program, because every early case may be a clue to a new localized outbreak, and sometimes a surprising number of infected individuals can be unearthed by adequate follow-up methods. This group of papers is most worthy of careful study by those interested in the problem.

It is worth mentioning that the fifth and sixth supplements[10] of *Venereal Disease Information* are a useful help to the general practitioner in the handling of syphilis. These concise booklets should be regarded merely as a guide and not as a thorough dissertation on the subject matter.

In any attempt to develop statutory control of syphilis there are three essential features: first, an adequate educational campaign over a period of years to obtain the co-operation of physicians and the public; second, a preservation of the opportunity for individual medical interpretation by properly qualified physicians of the complex problems which arise; and third, the insistence on an adequate physical examination as well as on laboratory procedures.

### EXPERIMENTAL RESEARCH

Perhaps the one outstanding advance of the year is the work of Eagle[11] on the in vitro effect of arsenic and bismuth compounds on *Treponema pallidum*. Years ago unsuccessful attempts had been made along this line. Eagle's success opens many vistas of perhaps startling advances. Time-consuming animal experiments on the therapeutic activity of various drugs may possibly be replaced. More accurate information on the mode of action of drugs may be obtained. This work of Eagle has already been applied from the clinical standpoint by the successful use of arsphenaminized citrated blood in the prevention of transfusion syphilis.[12]

Turner and his associates[13] report that some

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†Assistant in dermatology and syphilology, Harvard Medical School; assistant in dermatology, Massachusetts General Hospital.

degree of protection is afforded by syphilitic human serum in experimental rabbit syphilis. This substantiates the occurrence of protective antibodies in the serum of human syphilitic patients. Becker[14] has done similar work, utilizing a flocculate obtained from syphilitic human serum, and has shown that this material is antigenic. Landsteiner's haptene theory offers the best explanation of the mechanism involved, in Becker's opinion.

The massive therapy method has been pursued still further. This method of treatment, while holding a great deal of hope and promise, cannot be accepted until ten or fifteen years' observation bears out the results of a so far maximal period of five years' observation. Several European investigators using the technic have reported a high incidence of reactions. In this country the work of Chargin and Hyman[15] and their collaborators seems to have been most carefully carried out, and their results are more satisfactory than others reported, but complications are undesirably high in number. It should be emphasized that this type of therapy is still in an experimental stage. It is unsuitable for mass application and probably always will be.

### Diagnosis

An advance in the laboratory diagnosis of syphilis has been made in the development by Krajian[16] of a staining method for *Treponema pallidum* in tissue sections. This stain for smears containing the organism had been previously reported and has had a wide clinical application. The frozen-section method has some advantages over the dark-field and smear-stain methods, but it involves a delicate technic.

### Serology

There has been further search for a more rapid serological technic. Many reports[17] have appeared concerning the use of the Laughlen test for syphilis, which is comparatively new and rapid, but there is considerable disagreement in the reports. This method deserves further study.

Standard procedures should be employed until there has been wide experience with new methods. Comparative studies of the merits of already established serological procedures continue to be reported. The value of routine employment of a very sensitive flocculation or precipitation test, checked by a less sensitive but sometimes more specific complement-fixation method, has been emphasized by Tuft and Richter[18] in reviewing the Kline, Kahn and Kolmer methods.

Clinical confirmation of various serological procedures has been subjected to further study. Crawford and Ray[19] have reviewed a large series of serological discrepancies, and point out that a persistently positive Hinton test, even though unaccompanied by a positive Wassermann reaction, may be considered diagnostic of syphilis. When exhaustively studied 50 per cent of the cases in this large group were found to exhibit other evidence of syphilis. Serological discrepancies are continually arising and need further extensive study. It is important that in questionable cases there be repeated retesting of blood by several methods in different laboratories and searching physical examination. The question of false positives arises, but the temporary occurrence of a reagin-like substance has rarely been found in the blood of some otherwise normal individuals.[20] Various bacterial diseases other than syphilis and certain noninfectious diseases may give rise to this situation.[21] Infectious mononucleosis has been found to give an occasional false positive.[22] Further evidence has appeared which verifies previous assertions that malarial infections may cause a temporarily positive Wassermann or Kahn reaction.[23] It is possible that a solution of these enigmas lies in a more extensive application of quantitative serological methods. Kahn[24] cites many advantages of such a procedure. It is to be emphasized that an ordinary qualitative test reported as "++++" means nothing except that it is positive, giving no quantitative information.

Whether a negative Hinton test rules out neurosyphilis in the absence of clinical evidence and thus obviates the necessity of spinal puncture is still a controversial point according to Hazen,[25] who states that from 15 to 20 per cent of cases of neurosyphilis will be found to have a negative blood serological test even with the most delicate methods. The Davies-Hinton reaction performed on spinal fluid is reported by Marquis[26] and Brenner and Merritt[27] to have a higher degree of specificity and sensitivity than has the Wassermann test. Further study is indicated, and meanwhile both tests should be continued on spinal-fluid specimens.

### Clinical Problems

Late asymptomatic syphilis has been emphasized by a number of studies during the past year. The prognosis of the so-called latent group was reviewed by Morgan,[28] who found that life expectancy is shortened approximately 17 per cent in these patients. The amount of activity found to occur subsequent to the diagnosis of latent syphilis was said by this author to be 25 to 35 per cent. Adequate treatment of this group of syphilitic patients reduces the morbidity to 5 per cent. Even if the figures quoted by Morgan are a little higher than might be expected by some

authorities, they should serve as a warning to those who would summarily dismiss asymptomatic late syphilis. O'Leary[29] in a review of latent syphilis emphasizes the fact that adequate treatment in the early stages would prevent the appearance of incapacitating or death-dealing complications in 98 per cent of patients. The present incidence of 60 per cent asymptomatic syphilis would thus be theoretically reduced to 2 per cent. The point should be brought out here that the continuation of O'Leary's "permanent latency" will not be maintained by the patient's self-generated resistance in some cases. A short course of bismuth once or twice yearly will obviate trouble in practically all cases.

Beckh and Kulchar[30] state that too long usage of any one drug may be partially to blame for resistance to treatment. They also found that the simultaneous use of two drugs seems to contribute to treatment resistance, perhaps owing to underdosage with the arsphenamines.

### Syphilis and Tuberculosis

A number of articles have appeared in regard to coexistent syphilis and pulmonary tuberculosis. An analysis of the more recent reports seems to indicate that the two diseases can occur in the same patient without undue exaggeration of either, except in the Negro. Patients with pulmonary tuberculosis appear to tolerate antisyphilitic therapy as well as nontuberculous individuals. The effect of tuberculosis on the serological reactions for syphilis has been a rather controversial point for some years. Parran and Emerson[31] state that tuberculous toxemia may contribute a confusing factor to the serological study of syphilis, but should not present a major problem in the clinical interpretation of results obtained with carefully conducted serodiagnostic procedures.

### Cardiovascular Syphilis

Recent studies indicate a gradual diminution in cardiovascular syphilis. Welty[32] reports the clinical and pathological records of 15,000 consecutive postmortems over a ten-year period. His figures bear out the impression of both the decrease in incidence of cardiovascular syphilis and its lessening importance as a cause of death. A study by Thompson, Comeau and White[33] of syphilis of fifteen to twenty-five years' duration revealed only 7 per cent of cases as having definitely demonstrable cardiovascular syphilis. None of the cases having had even a minimum of adequate therapy were among the 7 per cent with cardiac damage. This supports the value of therapy in the prevention of cardiac syphilis.

### Neurosyphilis

A concise review of the treatment of neurosyphilis by Merritt[34] is a partial guide for the general practitioner. Kopp and Solomon[35] studied the malarial treatment of 302 cases of general paresis. The best clinical results were obtained in those patients who received one hundred and fifty hours or more of temperature exceeding 100°F. rather than extremely high temperatures. Patients receiving more than ten paroxysms obtained better clinical results than did those with less.

Observations indicate that the prognosis in paralytica dementia is strongly influenced by the personality pattern. The schizophrenic type should be viewed as of very grave prognostic significance.[36] Patients in the delirious, apathetic and agitated groups have the best prognosis, especially those with a well-integrated preparalytic personality.[37]

Thiamin chloride has been found useful for certain cases of lightning pains in tabes dorsalis. Metildi[38] treated a small series of cases with parenteral injections of this drug at weekly intervals. It has been our experience, however, and that of O'Leary[39] that very large daily intravenous doses are usually necessary to produce results.

### Syphilis in Pregnancy

A rather harsh criticism of the routine therapy generally accepted for pregnant syphilitic women has been made by Ingraham,[40] who questions so much active arsenical therapy during pregnancy. Naturally any physician will give full consideration to the physical condition of the individual woman patient in planning treatment for the remainder of the pregnancy, and will weigh carefully the risks of treatment and the possibility of a syphilitic child. Moore[41] in commenting on this paper points out that bismuth alone has not succeeded in preventing congenital syphilis, and that the abandonment of arsenical therapy during pregnancy would most certainly increase the incidence of congenital syphilis.

### Congenital Syphilis

A most excellent treatise on the diagnosis of congenital syphilis by Black[42] is well worth reading in toto. Clear tables illustrate the essential pathognomonic criteria. The vital necessity of diagnosing congenital syphilis at the earliest possible time in life and the institution of effective treatment as soon as the diagnosis is established are emphasized. No treatment should be given without an absolute diagnosis, and if the child of a syphilitic mother gives negative reactions the follow-up should continue over a period of years

in all cases. Offspring of syphilitic women may exhibit no diagnostic evidence of any sort until the age of five years or later. This author also strongly advises lumbar punctures in all congenital syphilitic patients. Hinsie[43] reports on neurosyphilis in children, and quotes the striking incidence of 33 per cent positive spinal-fluid findings when routine lumbar punctures were done on congenital syphilitic patients with positive serological blood tests.

The ocular manifestations of congenital syphilis are reviewed by Kahn[44] and McLeod.[45] The former states that the eye is more frequently affected than is any other organ in congenital syphilis and that there is a higher percentage of ocular lesions than of positive blood serological findings. A report by Klauder and Cowen[46] on corneal examination by slit-lamp microscopy in the diagnosis of late congenital syphilis is of considerable interest. Its use is essentially restricted to ophthalmologists.

Several reports on the treatment of congenital syphilis bear out the importance of full therapeutic doses in accordance with weight rather than age, and the usefulness of therapy even in older congenital syphilitic patients. Acetarsone is not finding as much favor as formerly; later reports indicate less satisfactory results and higher morbidity, due to complications and reactions, than was originally hoped.

## Therapy

Although new preparations of arsenic continue to be studied, it seems that bismuth is at present in the foreground of the investigative field. Reports on toxicity, absorption, excretion, retention and therapeutic activity coincide with previously accepted knowledge of the various bismuth compounds. A simplified technic for administering old arsphenamine is reported by Cannon.[47] It is useful for those employing this drug in office practice. A similar technic has been used for many years at the Massachusetts General Hospital.

Acetarsone has been studied further by Pillsbury and Perlman.[48] The effect of this drug in arresting congenital syphilis is inferior to arsphenamine and bismuth preparations, and the incidence of reactions is high. This drug cannot be experimentally controlled in regard to toxicity or effectiveness as can the other arsenicals. One cannot be sure that patients are taking their medication as directed. This objection applies to any oral treatment of syphilis. Robinson and Robinson[49] report that Acetarsone is a dangerous drug. They state that it is definitely contraindicated for adults and also issue a warning about self-medication.

Several papers have appeared in regard to the simultaneous use of arsenic and bismuth compounds. The bulk of opinion seems to be against this combined treatment. Disadvantages include higher risk of damage to the patient, slower reversal of serological tests[50] and a higher incidence of relapse. Beckh and Barnett[51] support this attitude. They report almost four times as high a percentage of mucocutaneous relapses among cases treated with arsenic and bismuth simultaneously. The alternating continuous system therefore seems to be preferable.

Mention was made in last year's report of oral bismuth therapy in syphilis. Work along that line has been continued, and very recently further reports of the clinical application have appeared. It is believed that considerable caution should be exercised in the application of any oral treatment for syphilis. It will always be difficult, even with the most co-operative patient, to be certain that the prescribed dose of oral medication has been received. The patient will tend to stray farther from the influence of the physician, thus reducing the probability of co-operation. In the place where such therapy would be of the greatest economic value, namely the indigent population, oral medication would be without doubt the most abused. The results would be an inevitable increase in relapse and spread of the disease. We cannot know for many years the ultimate effect of any new type of medication for syphilis. A most serious danger to the public health is the possibility of self-treatment which oral therapy for syphilis is bound to encourage. Oral medication should be restricted to certain very narrow limitations, which might include patients unable to secure regular treatment by a physician, the occasional unusual patient who cannot tolerate intramuscular injections, perhaps some cases of cardiovascular syphilis and a few selected cases of congenital syphilis. A recent editorial[52] gives a concise summary of this situation.

## Treatment Reactions

"Erythema of the ninth day" has received considerable attention. It is now relatively well accepted[53, 54] that this reaction is not a contraindication to the continuance of intravenous arsenicals, but a temporary reduction in the dosage is advocated. Complications of the ninth-day erythema, such as jaundice, prolonged dermatitis or hematologic disturbances, are a definite contraindication to arsenicals, at least for a considerable time after recovery. It is essential to differentiate this eruption from the early stages of exfoliative dermatitis. One case has been reported in association with bismuth therapy.[55] It has also occurred following gold injections.

Gastric intolerance to the arsphenamines is discussed by Irgang[56] in a lengthy but interesting treatise. One warning seems essentially worth while. He states that any arsenical hepatitis not responding to three weeks of routine detoxication therapy should be watched with exceptional care, as this may presage yellow atrophy of the liver.

Post-arsphenamine jaundice was studied by Lane[57] in a review of 100 cases. The four factors of causal importance in this series were arsenic, syphilis, bacterial infections and bismuth. Bismuth appeared to be a precipitating factor to a larger degree than is usually expected. Soffer[58] reviewed all the reports of jaundice in the available literature. He found that 1 per cent of syphilitic patients develop jaundice. From 1 to 14 per cent of those having jaundice were reported to have developed yellow atrophy of the liver. This is a disturbing percentage. Cirrhosis of the liver may play a greater part than is generally realized. The use of cholagogues and choleretics is held to be an unsound form of therapy.

Tryparsamide reactions have lately been reported. These include numerous nitritoid reactions, occasional dermatitis and a case of acute yellow atrophy of the liver. This drug until relatively recently was not known to cause any reaction other than damage to the optic nerve. A detailed and comprehensive review of the literature on tryparsamide was recently published by Hinrichsen.[59] It is assumed that there may have been an unnoticed deviation in the method of manufacture or possibly an unintended alteration in the basic materials used. These matters are being investigated.

Optic nerve damage from tryparsamide is being investigated by Muncy,[60] and he reports that the vitamin B complex seems to act as a preventive. This author has also used the vitamin B complex as a curative agent after tryparsamide damage has begun to appear; large doses should be used in these cases. Muncy believes that preparatory bolstering of the patient with the vitamin B complex before the use of tryparsamide is of considerable value.

Astrachan[61] indicates that the administration of liver extract seems to help in preventing reactions of intolerance to arsenicals. It may be of some help in hastening recovery from the aftereffects of arsenic intolerance.

* * *

That definite progress is being made in the control of syphilis is indicated in a recent release of the United States Public Health Service.[62] Not only have the facilities for diagnosis and treatment been greatly enlarged, but 60 per cent more persons have been brought under treatment than in the previous twelve months. Approximately 103,000 patients were discharged from clinics as cured or arrested, as compared with 78,000 in 1938. Such figures lend encouragement to the campaign against syphilis.

416 Marlboro Street.

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# CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

## CASE 26061

### PRESENTATION OF CASE

A sixty-year-old Italian barber was admitted complaining of severe epigastric pain.

Twenty years before he had been examined at an outside hospital for the question of peptic ulcer, but the diagnosis had not been established. He was well from then until six months before entry, when he began to complain of indigestion characterized by a sense of epigastric fullness. Two weeks before entry this symptom had become worse and for the first time he began to notice vague epigastric pains. Fourteen hours before being seen he had a sudden onset of severe, prostrating, epigastric pain. He vomited a small amount of material described as resembling coffee-grounds. His local physician was called and gave him some medicine in water to drink. This produced great accentuation of the abdominal pain. The doctor was called back again four hours later, at which time the patient was found in collapse. There was definite cyanosis, dyspnea and a weak thready pulse, and the blood pressure could not be obtained. The abdomen was somewhat distended, and was tender, particularly in the upper half; there was moderate spasm. The patient was referred to the hospital after he had been given two ¼-gr. doses of morphine.

Physical examination showed a well-developed and nourished man, who was cyanotic, with gasping respirations and a weak and thready radial pulse, markedly dehydrated and too ill to respond to questions. The heart sounds appeared normal, at about the rate of 110 beats a minute. The chest was clear to auscultation except for a few moist rales. The abdomen was moderately distended throughout. There was diffuse tenderness, most evident in the epigastrium and under the left costal margin. There was definite spasm of the entire epigastrium. Auscultation revealed no audible peristalsis. The flanks bulged somewhat, giving one the impression of fluid in the peritoneal cavity; the liver dullness was not obliterated. Rectal examination was negative. There was slight pitting edema of both ankles. The blood pressure was 60 systolic, 40 diastolic.

The temperature was 101°F.

The blood showed a red-cell count of 4,100,000 with 110 per cent hemoglobin, a white-cell count of 7400, and a normal differential.

X-ray examination in sitting posture revealed no air under the diaphragm and gave evidence of intestinal ileus because of one slightly dilated loop of small bowel containing a fluid level. The roentgenologist suggested that the radiance of the film was evidence of free fluid in the abdomen. There was also evidence of some fluid in the left pleural cavity. The patient's condition contraindicated exploration, particularly as no diagnosis could be arrived at. He was given a transfusion and Ochsnerized. Four hours later, examination showed the patient's condition to be somewhat worse. The abdomen was more distended and more spastic. The chest signs had changed remarkably, there being complete immobility of the left chest, with flatness to percussion and no breath sounds. Bronchial breathing, with increased fremitus, was heard at the right base. A chest tap through the left eighth interspace yielded a large amount of dirty brown fluid, which was odorless and was shown on smear to contain red blood cells in various stages of degeneration and amorphous yellow material presumed to be hemoglobin; a rare gram-positive diplococcus was seen. Six hours later the patient died.

### DIFFERENTIAL DIAGNOSIS

DR. TRACY B. MALLORY: Once in a long interval it seems only fair to turn the tables and today they have given me a case to discuss from the clinical point of view. After reading the history I wondered why this case was chosen for me and tried to decide whether it was to be regarded as a manifestation of touching faith on the part of my colleagues or of a vindictiveness that I hate to contemplate.

We are faced here with the paradox of a man with a perfectly clear story of an abdominal catastrophe whose only unequivocal clinical findings seem to be in the thorax. If we take the story alone we have a man, who previously had been in good health except for the vaguest of prodromal symptoms, suddenly developing prostrating abdominal pain, who in a period of a few hours goes into shock. That immediately makes one think of a perforated viscus, possibly of acute hemorrhagic pancreatitis or of mesenteric thrombosis, — although the pain is repeatedly described as epigastric rather than abdominal, — and conceivably of aneurysm, although there is no mention of pain in the back. With the old history of a question of ulcer twenty years ago, with the vomiting of some material described as coffee-grounds, — we wish we could rely on that and that we had some objective evidence to confirm it, — one would

think first in terms of peptic ulcer, and strongly suspect perforation. We look for evidence of the development of general peritonitis and get suggestive evidence of it, but the signs and symptoms all remain localized in the upper part of the abdomen and in the epigastrium. Now sometimes clinical signs of peritonitis such as this indicate a peritonitis limited to the lesser omental cavity, and an ulcer might easily perforate into that, and he might not yet have a general peritonitis.

The question comes up whether this is true shock or possibly response to hemorrhage. It sounds to me rather more like a picture of shock. The cyanosis fits that better. The constantly maintained low blood pressure sounds like shock, as does the fact that several hours later he still had a hemoglobin of 110. His blood was apparently concentrating rather than showing evidence of anemia, which makes shock more likely than hemorrhage.

I cannot, however, think of any ordinary complication of gastric ulcer that could produce what we find later. We have the statement that when he was first seen the thorax was negative to auscultation. I think we probably have to take that with a grain of salt. The examiner was probably thinking of an abdominal condition, as I thought when I read to that point, and I do not rely on that observation. Moreover, at the time he came to the x-ray department they had already found that there was something in the thorax. On the next clinical examination the breath sounds were diminished. Soon they disappeared completely over the left chest, which became fixed, no longer showed respiratory movements and was flat to percussion. Eventually they tapped it and got off material that we are led to believe was indicative of old hemorrhage.

So we have to consider the possible thoracic lesions, and there are several, which produce the picture of shock and pain localized in the abdomen. Coronary thrombosis can certainly do it, but coronary thrombosis could not account for the fluid which was withdrawn on chest tap unless there was also a rupture of the heart and the pericardium was so distended that they succeeded in tapping it. This material which was withdrawn, if we are right in our assumption that it is blood showing evidence of disintegrative changes, must have been acted on by ferments in order to change its consistence and color. Blood in a serous cavity does not undergo this change for days, so that the assumption of a ruptured heart would force us to assume that the disease is of much longer duration than the history would indicate. That does not seem a satisfactory explanation. Infarcts of the heart do not perforate for a number of days, — four, in my experience, is about the minimal time for them to occur, — and we have nothing to suggest that anything so severe as infarction of the heart occurred four days before the present illness. So I am inclined to rule out infarct of the heart and hemopericardium. Pulmonary embolus can produce shock. The story does not sound very much like it, but it can produce abdominal pain and it can produce pleural effusion which can be bloody, but again the time is too short for this to have become old, changed blood.

This seems to eliminate most of our possibilities except for the organs which pass from the thoracic into the abdominal cavity, and one certainly must think of the aorta. It is quite possible, I think, for a rupture of the aorta in the very low thoracic or upper abdominal region, bleeding perhaps into the leaves of the diaphragm, to result in blood in both the abdominal and pleural cavities simultaneously or one shortly after the other. We are without benefit of the results of serological tests, and sixty is a little late for a luetic aneurysm. So if it is rupture of the aorta, it is probably one due to senile ectasia or a deep atheromatous ulcer rather than to a syphilitic aneurysm.

There is one other possibility which certainly comes to mind. Every now and then the stomach is not in the abdominal cavity but in the thoracic cavity. That would explain most of the symptoms here. I think we should still have to assume a rupture of the stomach; the extravasation of material into the hernial sac would completely fill the pleural cavity and would also probably extend down into the lesser omental sac and account for the suggestive signs of peritonitis that we have there. Though that seems like a fanciful hypothesis, it would be my first guess. If there are some x-ray plates I should like to have Dr. Hampton show them before I finally commit myself.

Is that a fluid level on the left side?

DR. AUBREY O. HAMPTON: Yes.

DR. MALLORY: How high?

DR. HAMPTON: At the eleventh interspace posteriorly. It is more the shape of the fundus of the stomach than it is that of the small bowel.

DR. MALLORY: Can you tell where the level of the diaphragm is?

DR. HAMPTON: No. At the time of the x-ray examination he had extensive density in the left side of the chest, which might have influenced the physical examination somewhat later on. He was turned on the left side, and the film was taken to show free air but none was seen. I do not believe the radiologist was justified in calling this a loop of the small bowel; its interpretation is

the most important thing. The fundus of the stomach is probably below the diaphragm.

DR. MALLORY: I should probably listen to Dr. Hampton but the quality of the plates is certainly very poor, and I will stick to my two previous diagnoses. I will put diaphragmatic hernia with rupture of a thoracic stomach first, and aneurysm as the second possibility.

DR. HENRY NIGRO: I took care of this man, and I might say that when the aspiration was done the fluid did not look like anything that is often seen. It was neither pus nor clear fluid. They did examine a stained smear, and a couple of gram-positive diplococci were found. Dr. Chapman saw him a few hours later, and after looking at this material, which had a dirty brown appearance, he thought of testing it with Töpfer's reagent. When that was complete it was evident that the fluid was gastric contents.

DR. MALLORY: I meant to say that I wished this material, described as odorless, had been sniffed by Dr. Richardson or someone in whose nasal sensorium I have complete confidence.

DR. GRANTLEY W. TAYLOR: Why would not carcinoma of the upper part of the stomach with perforation both ways account for the findings?

DR. MALLORY: I think that is a good idea and should have been suggested.

DR. ALLEN G. BRAILEY: I should think perforation of a peptic ulcer through the diaphragm might be possible.

DR. FRANCIS T. HUNTER: I should like to emphasize the fact that I do not know of any way of turning hemoglobin into "amorphous yellow material." That description is misleading.

### CLINICAL DIAGNOSIS

Rupture of peptic ulcer of esophagus.

### DR. MALLORY'S DIAGNOSIS

Rupture of intrathoracic stomach.

### ANATOMICAL DIAGNOSIS

Rupture of peptic ulcer of esophagus.

### PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: Here is the specimen that was removed at autopsy (Fig 1). There was no diaphragmatic hernia, but what had occurred was a perforation of a peptic ulcer of the lower end of the esophagus. It had perforated into the left pleural cavity, had dissected posteriorly and retroperitoneally down to the abdomen for a short distance and terminally into the peritoneal cavity. There were 2000 cc. of fluid in the left pleural cavity. Unfortunately, we have not yet been able to get microscopic sections of the ulcer but it is most likely a peptic ulcer arising in ectopic gastric mucosa in the esophagus. This is the usual cause of these ulcers, although simple autodigestion is sometimes given as the explanation. Since peptic ulceration has a characteristic fibrinoid base it should be quite easy to recognize.

FIGURE 1. *Perforated Peptic Ulcer of Esophagus.*

DR. EDWARD HAMLIN, JR.: I should like to emphasize the sequence of events. The chest was essentially negative when the patient was first seen. Apparently what happened was that the mediastinal mass dissected retroperitoneally first, thus causing abdominal symptoms, and a few hours later ruptured into the left chest. The sequence of events was quite startling to say the least. So far as I can tell from the clinical and the pathological records there has been only one other esophageal perforation. That was the case of Dr. Richardson's that was discussed at one of these conferences several years ago. It is apparently a relatively uncommon catastrophe. More often the ulcer penetrates into the aorta and the patient dies of hemorrhage.

DR. EARLE M. CHAPMAN: I saw this patient, and while discussing the case with the house officer, after having been told all the clinical picture, he said: "If this is an ulcer on the lower end of the esophagus that perforated, this fluid might be gastric contents." I thought it was an excellent idea, and so we did a gastric analysis and found combined acid present. We then made the diagnosis. Unfortunately, Dr. Mallory did not have that information beforehand.

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## CASE 26062

### PRESENTATION OF CASE

A thirty-three-year-old Italian laborer was admitted complaining of pain in the epigastrium.

Five years before admission the patient noticed a lump in the right pectoral region which gradually grew larger and finally painful. Two years before entry the mass was removed in an outside hospital. The pathological report, checked at this hospital, was "lymphosarcoma, lymphocytic type." Because pain persisted in the operative site the patient was referred to the Tumor Clinic where, during an eighteen-month interval, he was given roentgen therapy over the region of the axilla. The pain was relieved, and a few smaller palpable nodes in the area disappeared. He received only 1200 r in all. Throughout this interval he remained in excellent general health and was able to continue work. During frequent follow-up visits his chest remained clear and his physical examination negative, except for the finding of a small nodule in each axilla when last seen three months before admission. One month before entry, however, he felt a mass in the epigastrium after urination. Fourteen days before admission his house caught afire; he narrowly escaped death as he leaped down a flight of stairs to avoid the conflagration. He was excessively worried because of the loss of all his possessions, and two days later grew aware of a burning epigastric pain occurring daily before breakfast, relieved by drinking coffee and recurring a few hours after the evening meal when it was alleviated by "soda water." Throughout this latter illness he experienced no vomiting, weight loss, anorexia or black or tarry stools. Except for the fact that his brother had "ulcers," the patient's family, marital and past histories were negative.

Physical examination revealed a well-developed and nourished man who lay flat and quietly in bed. He was pale and complained of pain in the epigastrium and rectum. There were pea-sized shotty enlargements of the left axillary, the epitrochlear and the inguinal lymph nodes. The heart and lungs were normal. The entire epigastrium was tender, but exquisitely so in the mid-epigastrium. On inspiration a hard, round-edged spleen was felt to descend two to three finger-breadths below the costal margin. There was a painful, tender rectal sphincter, but no fissure or other abnormality was observed to explain this finding. The blood pressure was 125 systolic, 60 diastolic.

The temperature was 100.6°F., the pulse 100, and the respirations 24.

Examination of the blood showed a red-cell count of 4,200,000 with 72 per cent hemoglobin, and a white-cell count of 4200 with 74 per cent polymorphonuclears; the smear was essentially negative. A stool specimen showed a + guaiac test. Gastric analyses, both before and after histamine, revealed no free acid and a total acid of only 5 units.

A gastrointestinal x-ray series showed that the stomach was displaced to the right by a markedly enlarged spleen. The folds of the stomach, particularly those of the body close to the lesser curvature, were thickened. The remainder of the organ was negative. The duodenal cap showed no evidence of disease. There was no evidence of ulcer. Films of the chest showed a high diaphragm, particularly on the left, apparently due to an enlarged spleen. There was no evidence of intrapulmonary disease.

The patient ran an elevated temperature, which ranged between 100 and 103°F., throughout his seven-day hospital stay. Four days after admission he suddenly developed a sharp, stabbing pain with exquisite tenderness in the left upper quadrant. The constant, mid-epigastric distress and tenderness remained unchanged, however. The white-cell count rose from 4000 to 7000. At 9:00 p.m., on the seventh hospital day, he was seized with another severe, now mid-epigastric, "red-hot-poker-like" pain, distinct from that which remained in the left upper quadrant. He vomited a mouthful of fresh bright-red blood and continued to retch, with much nausea, until he had brought up 400 cc. The patient was given morphine sulfate (¼ gr., repeated fifteen minutes later) and a few swallows of sodium bicarbonate solution, both of which brought relief from vomiting and a diminution in pain. He sweated profusely, and the pulse became thin and irregular, at 150 beats per minute. One hour after the episode had started, his respirations became rapid, irregular, deep and gasping. This change became increasingly more pronounced, and a half hour later, or one and a half hours after the onset, respirations ceased.

DIFFERENTIAL DIAGNOSIS

DR. TRACY B. MALLORY: I might say that the x-ray treatment was given here at this hospital. The original biopsy was done outside, but we saw the slides.

DR. ROBERT R. LINTON: So you confirmed this diagnosis?

DR. MALLORY: We apparently did.

DR. LINTON: May I see the x-ray films?

DR. GEORGE W. HOLMES: Normally the diaphragm is a little lower on the left than on the right. Here it is higher, and the space between the fundus of the stomach and the diaphragm is increased; all of which confirms the statement in the text that he had a mass in the left upper quadrant, probably an enlarged spleen. In the stomach there is a peculiar appearance of the mucosal pattern, extending from the cardiac orifice down along the lesser curvature. I should think that was definitely abnormal. One would like to have had further observations. Evidently the man who did the examination thought the appearance was abnormal and described it as a prominent fold. From these films I cannot say anything very definite about the pyloric end of the stomach. The sphincter looks all right; the cap looks all right. What could it be? Lymphoma could produce prominent mucosal folds and nothing else. That is a possibility. Gastritis could do it — local gastritis. I think varices in the stomach could do it. We have never seen, or at least I have never seen, varices in the stomach; we have looked for them, and we have had some cases that at autopsy were proved to have had them as a cause of hemorrhage. But if one did see them I should think they would look something like this.

DR. LINTON: Was the esophagus examined for varices?

DR. HOLMES: They do not state whether it was or not, but it is a routine procedure in our clinic.

DR. LINTON: We have a man of thirty-three years of age who was admitted to the hospital because of pain in the epigastrium. Seven days after he was admitted he suddenly died, and it is up to me to explain the cause of his death. I believe that his exitus was due to hemorrhage, and I have to discover the origin of this hemorrhage. In reading over the history and physical examination, he presents two outstanding findings — hematemesis and splenomegaly. I am a little uncertain how much emphasis to place on the diagnosis of lymphosarcoma because the mass was a little unusual in location, especially without any other enlargement of the lymph nodes. By x-ray there is no enlargement of the mediastinal glands, and I rather think that the treatment with 1200 r was a small dose. Is that right, Dr. Holmes?

DR. HOLMES: Not if the diagnosis was correct.

DR. LINTON: The mass that he noted in the epigastrium after urination I should gather was most likely the spleen that was felt on physical examination. The laboratory studies are not helpful. I should like to know whether a Hinton test was done. Since it was not included here, it probably is of no significance; but it is one of the possibilities that has to be considered. The gastric analysis is of interest in that he had no free hydrochloric acid even after histamine.

In reviewing the case we must consider the causes of hematemesis. I think that one can rule out any esophageal cause of hematemesis, such as varices, mediastinal growth or perhaps an aneurysm rupturing into the esophagus, because of the lack of evidence by x-ray and physical examination of disease in these structures. Passing on to the stomach and duodenum we have to consider ulcer and carcinoma, which I think are pretty well ruled out by the x-ray examination. There is also the possibility he had an abdominal aneurysm with rupture into the stomach, but there is no record that there was an expansile pulsation in the abdomen, so we can rule out that possibility. We do have an x-ray diagnosis of gastritis, and it is possible to have a severe degree of hematemesis from gastritis, so that I think that is a likely possibility — one which we should consider seriously. Another important cause of hematemesis is portal cirrhosis with splenomegaly, but I am not aware that one can get hematemesis from portal cirrhosis without esophageal varices. There are other rare conditions, such as the splenic-vein thrombosis of Banti's disease, but again esophageal varices are necessary to explain hematemesis. There are certain other diseases, such as a primary blood dyscrasia, in which hematemesis is a possibility, but they are ruled out by the negative laboratory findings. I believe Hodgkin's disease and the various other forms of lymphoma are conditions which may cause hematemesis, though I am uncertain as to the mechanism. Syphilis is certainly to be considered in view of the fact that he had multiple, enlarged lymph nodes.

Did you find any record of the Hinton test, Dr. Means?

DR. J. H. MEANS: This is very disturbing. The blood was taken, but the Pathology Laboratory did not report it!

DR. LINTON: We have a young man with an enlarged spleen who had a sudden exitus from hemorrhage. I do not see how one can get away from the pathological report of lymphoma. It

is disturbing to me that there is no evidence of the disease on his admission to the hospital. I do not know whether splenomegaly alone can produce gastritis, but certainly gastritis can produce hematemesis, so I think we have to consider these two conditions, namely lymphoma and gastritis. Any other diagnosis would be mere guessing. In conclusion, since he had an enlarged spleen and a diagnosis of lymphosarcoma had been made on the mass removed from his right axilla, I must make lymphoma my primary diagnosis; I believe his bleeding came from the stomach and was most likely due to an acute gastritis.

Dr. Wyman Richardson: I think you have thrown out the diagnosis of thrombosis of the splenic vein too hastily. Why should he not have obstruction due to that or cirrhosis, and splenomegaly with gastric varices and hemorrhage from them?

Dr. Linton: I think that is a possibility. The unusual thing to me about the whole affair from this point of view is that he had such severe pain with hematemesis. When a man has hematemesis from ruptured esophageal varices he usually does not have much pain. It was chiefly because of this I thought that his bleeding was not from varices.

Dr. Edward B. Benedict: Why had he not had perforation of the stomach?

Dr. Linton: You do not see hemorrhage and perforation together.

Dr. Benedict: Not usually, but it is possible.

Dr. Linton: Yes, but I have never happened to see it.

Dr. Francis T. Hunter: I never saw this man, but I do not believe there is much question but that he had lymphoma with infiltration of the stomach and probably a ruptured stomach and a ruptured spleen. He must have had a hole through the spleen, which was already infarcted, according to the story. I think you are going to find a lot of lesions in the left upper quadrant.

Dr. Linton: An infarct is a good suggestion. Yet the white count is not in favor of lymphoma.

Dr. Hunter: In lymphoma the white count can be anything.

### Clinical Diagnoses

Lymphoblastoma, lymphocytic type.
Hematemesis.

### Dr. Linton's Diagnoses

Lymphoma.
Gastritis.
Hematemesis (from the stomach).

### Anatomical Diagnoses

Hodgkin's disease of spleen and retroperitoneal tissues.
Infarction and ulceration of the spleen.
Perforation of the stomach.
Fatal hemorrhage.

### Pathological Discussion

Dr. Mallory: The postmortem examination showed a tremendously dilated stomach which was completely filled by a cast of blood clot over a foot in length. Unquestionably the hemorrhage was massive enough to be the immediate cause of death. On the lateral wall of the stomach near the cardia was a large jagged perforation which led into a corresponding hole in the spleen, which was several centimeters in depth. The spleen was greatly enlarged, weighing 1700 gm., and showed tumor nodules and multiple infarcts. Beneath the spleen in the region of the tail of the pancreas and throughout the retroperitoneal tissues was extensive tumor, and this had infiltrated and completely occluded the splenic artery so that there was good reason for the infarction of the spleen. The mass had also infiltrated down as far as the left kidney, which showed some invasion of the peripheral portion. Unquestionably the source of the terminal hemorrhage was the perforation of the stomach and bleeding from the spleen itself. What the mechanism of the perforation was, I feel less certain about. In the face of completely absent hydrochloric acid it is difficult to believe that it could have been a peptic ulcer, and the histology does not suggest it. I think it is more likely that the spleen became adherent to the stomach and that infarction of the spleen occurred and broke directly through the stomach wall. I should be inclined to trace it that way rather than from the stomach outward. We could discover no tumor in the stomach walls — only severe gastritis.

Dr Means: There is a story of possible trauma ten or twelve days before death when he fell downstairs. I wonder if he injured his spleen at that time. A lymphomatous spleen is very easily traumatized. I remember one man with a huge leukemic spleen who squeezed it so hard one day that it ruptured and he died of hemorrhage. I wonder if he had a hematoma in the spleen in close approximation to the stomach.

Dr. Mallory: I think that is possible. The lesion lay at the upper end of the spleen. The area was covered by the thoracic cage, however, so it could not be directly injured by trauma unless by contrecoup.

A Physician: Do we not have occasional

lymphomatous ulcers of the stomach with perforation and general peritonitis?

DR. MALLORY: We have seen lymphomatous infiltration of the stomach with perforation, but here there is minimal evidence of involvement of the stomach itself. From the slides I cannot be certain there is any. The most surprising thing from the histological point of view is that the lesion was quite characteristic of Hodgkin's disease and in retrospect it is hard to believe that we could have called it lymphosarcoma. I wonder if we were not misquoted.

DR. MAURICE FREMONT-SMITH: Why did he bleed so much if the splenic artery was obliterated?

DR. MALLORY: That is hard to answer, but massive hemorrhage is common following infarction, and the presumption that a good deal of it must have been retrograde from the veins seems to be the only explanation.

DR. GEORGE A. MARKS: Was the splenic vein involved?

DR. MALLORY: By pressure but not by direct tumor infiltration, as the artery was.

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## PHARMACOTHERAPY

The criticism is valid that, while clinical diagnosis has advanced remarkably in the past quarter of a century, progress in medical treatment is lagging. Pharmacology, the scientific basis of therapeutics, has come to be considered by many to be of little practical value, and a number of schools have gone so far as to abolish their pharmacological departments.

Pharmacology, as established by Schmiedeberg and his pupils, is essentially a science dealing with the effects of chemical and biologic agents on normal animals and on surviving organs. Due to such systematic investigations of old and new drugs, critique invaded therapeutics for the first time, with the result that the majority of previously used medications were eliminated as useless. But great though it was, this contribution of experimental pharmacology was to a large extent limited. Often unjustified conclusions regarding the treatment of disease in man were drawn from observations on normal animals. After a half century of this sort of analytic activity, it is natural that the field became somewhat sterile and ceased to draw able and inspired workers.

Recently, however, there has seemed to be justification for the optimistic belief that what is considered by many a vicious circle in the development of pharmacology is actually the beginning of the second circle of a spiral—a form that scientific progress often takes. Among the developments important to this expected renaissance of pharmacotherapy are the advances in quantitative chemical, physiological and immunological methods applicable to men, the increasing opportunities to induce disorders and diseases in animals and the close co-operation between groups of investigators in allied fields. These developments make it possible systematically to advance the findings of the laboratory to the treatment of diseased animals and, eventually, of men. As a result more important therapeutic discoveries have been made during the past decade than ever before—discoveries which have forever eliminated therapeutic nihilism in medicine. Progress in infectious diseases, in nutritional deficiencies, in dysfunction of the glands of internal secretion and in nervous disorders are but a few of the therapeutic triumphs.

It is essential to appreciate, however, that not pharmacological laboratories alone but all departments of physical, biological and clinical sciences have played their roles in these discoveries. For example, there are at present more than two hundred pharmacotherapeutic investigations in progress at Harvard University, scattered throughout its various scientific departments. Such widespread activity calls for helpfulness and coordination by a group representing the diversified interests involved. In the distant past, universities were institutions composed of groups of thinkers working in close association, and the

segregated departments of today are the result of the material and technical developments of the past century. But new interests that cut across a number of departments call once more for close personal association. It is significant, therefore, that President Conant has recently appointed a University Committee for Pharmacotherapy. It is hoped that, as a result of the activity of this committee, clinical and scientific interests will be brought closer together, pharmacotherapeutic investigations will be better guided, teaching of pharmacology and therapeutics will improve, postgraduate education in this diversified field will be fostered, and co-operation between the investigators in academic and industrial fields will be promoted.

Able students frequently enter surgery because they feel they can do more for the patient. The recent, rapid developments in pharmacotherapy should convince students that clinical medicine also offers great opportunities for treatment. Here there opens up a highly specialized field where physicians must use therapeutic agents which are often more effective and more dangerous than the scalpel. Pharmacotherapy may be considered by medical historians of the future as one of the most significant developments in the history of medicine in the twentieth century.

## PATHOLOGICAL SERVICE FOR THE SMALL HOSPITAL

With the increasing complexity of diagnostic procedures, the wider recognition of the advantages that the pathological laboratory can afford the clinician and the higher standards for approved hospitals, there has been an increased demand for pathologists. So far as the large centers are concerned, this can be readily met, but in the small and relatively isolated hospitals, it is a very definite problem. Rarely is the size of the hospital large enough to support a full-time pathologist, and about the only feasible plan has been the grouping of a number of hospitals together to utilize the services of a single pathologist.

Recognizing that a number of the small hospitals away from the large medical centers have difficulty in arranging for pathological service, the New England Pathological Society has established a committee with the hope of giving impartial advice to hospitals interested in establishing pathological laboratories and of bringing together competent men desiring positions and hospitals desiring pathologists. A letter in this issue of the *Journal* calls attention to the plan. The committee is now endeavoring to aid a group of three small hospitals with their laboratory problems.

Should the present plan work out successfully, it may point the way for a more effective utilization of the help that laboratory procedures can give in diagnosis and treatment.

## MEDICAL EPONYM

### Banti's Disease

Guido Banti (1852–1927), professor of pathological anatomy of the medical faculty of the University of Florence, Italy, published his first account of the disease which bears his name in *La semaine médicale* (**14**: 318, 1894). It is entitled "La splenomegalie avec cirrhose du foie." The original Italian draft of this article appeared a month later in *Lo sperimentale, sezione biologica* (**48**: 447–452, 1894), with due credit to the French journal. An English translation appeared in the English edition of *La semaine médicale* (**2**: 364, 1894). The following quotation is from the English version:

> In 1882, my attention was directed to the existence of a symptomatic and anatomo-pathological complexus which, so far as I am aware, has never been described, and which may well be considered as a special kind of disease, namely, *splenomegaly with cirrhosis of the liver*. . . .
>
> The symptoms of the disease may be divided into three groups, corresponding to as many periods, viz.: the *prae-ascitic* stage, the *ascitic* stage, and an *intermediate* stage.
>
> The symptoms of the *prae-ascitic* stage are: tumefaction of the spleen and anaemia. . . .
>
> In the cases with which I have had to deal, the prae-ascitic stage varied in duration from one year to four years and a half.
>
> The *intermediate* stage is characterized by the appearance of dyspnoea, intestinal disorders, and sometimes haemorrhoids. . . . This stage lasts a few months.
>
> In the *ascitic* stage a liquid effusion takes place into the peritoneum. . . . The anaemic symptoms become more and more marked; but the examination of the blood continues to give evidence of a normal number of leucocytes. This stage lasts from seven to eight months and ends in death.

## OBITUARY

### FREDERICK HENRY THOMPSON

1844 - 1939

In the December 21 issue of the *Journal* the death of Dr. Frederick H. Thompson, of Fitchburg, at the age of ninety-five years and four months, was recorded.

He had the distinction of being the oldest living alumnus of Harvard Medical School, and also the oldest living member of the Massachusetts Medical Society, having joined in 1870. Even after giving up active participation in the affairs of the Society in 1920, he kept in touch with local medical affairs during the later years of his life.

He was well qualified for medical practice by graduation from Harvard Medical School and an internship at the Massachusetts General Hospital. After the completion of his hospital training, Lancaster welcomed Dr. Thompson as the community doctor and physician to the Massachusetts Industrial School for Girls, where he established a reputation for sound practice.

His studies and service at the Massachusetts General Hospital under the direction of the prominent surgeons of that era had fostered an ambition to make surgery the major interest of his professional career, and in looking for greater opportunities, the growing city of Fitchburg presented attractive possibilities in this direction for which Lancaster could not compete; so after four years he moved to the larger community and soon became recognized as a valuable factor in the professional and civic life of that city. Practically all the important educational and cultural organizations of Fitchburg sought his support, which was freely given, although his major interest was always centered in medicine. Appointments to various official positions followed, among which were assistant surgeon and, later, surgeon of the 10th Regiment of the Massachusetts National Guard, secretary-treasurer and president of the local Society for Medical Improvement, orator, librarian and president of the Worcester North District Medical Society, corresponding secretary of the Massachusetts Medico-Legal Society, two appointments as medical examiner of the third district of Worcester County, member of the Board of Health, member of the United States Pension Examining Board, vice-president and councilor of the Massachusetts Medical Society and delegate to the House of Delegates of the American Medical Association in 1911 and 1912. During the World War, Dr. Thompson served as chairman of the Selection Service Board of the 14th Massachusetts District, as captain-surgeon of the Massachusetts National Guard and as a member of the Medical Reserve Corps. To this long list, other official city positions could be added.

Perhaps his longest and most concentrated interest was the Burbank Hospital. He was associated with its founding in 1891 and had served as a trustee and as a member of the staff, first as an active member and later as a surgical consultant. His influence in these positions was always of first importance in the growth of the hospital.

Here is presented a partial record of a busy and useful life of service which has inspired many tributes of affection and appreciation by his city and state. Those who were privileged to know him intimately profited by his sincerity and by his loyalty to high ideals and energy, which combined to establish qualities of leadership that brought success to his undertakings.

The history of Fitchburg will preserve the records of the accomplishments of this loyal and devoted citizen through all time, and his example will stand as an inspiration to those on whom his mantle has fallen.

W. P. B.

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## MASSACHUSETTS MEDICAL SOCIETY

### SHATTUCK LECTURE

The Shattuck Lecture will be given, as usual, following the annual dinner on May 21, and the Society has been extremely fortunate in obtaining Dr. Ernest W. Goodpasture as the speaker. The provisional title of the address is "Immunity to Virus Infections: Some theoretical and practical considerations," and Dr. Goodpasture is eminently qualified to discuss the subject, since for many years he has been an outstanding contributor to the various problems associated with virus infections.

From 1915 to 1922, Dr. Goodpasture was a member of the Department of Pathology at the Harvard Medical School, being assistant professor the last three years, and from 1922 to 1924 was director of the Singer Memorial Research Laboratory in Pittsburgh. In 1924 he was made professor of pathology at Vanderbilt University School of Medicine in Nashville, Tennessee, which position he now holds.

## SECTION OF OBSTETRICS AND GYNECOLOGY*

RAYMOND S. TITUS, M.D., *Secretary*
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Boston

### PUERPERAL SEPSIS FOLLOWING LOW-FORCEPS DELIVERY

Mrs. J. R., a thirty-four-year-old primipara, entered the hospital on July 6, 1914, when approximately thirty-seven weeks pregnant, because of a systolic blood pressure of 190, edema and a large trace of albumin in the urine. She started in labor spontaneously on July 16 subsequent to premature rupture of the membranes, and was delivered of a stillborn child on July 17 by a simple low-forceps operation.

The family history was not recorded. The patient had had the usual children's diseases and one rather serious period of melancholia which lasted six weeks. Catamenia were regular, having begun at fourteen, with a twenty-eight-day cycle. The last period was October 23, 1913, making the expected date of confinement August 3.

Physical examination at the beginning of pregnancy showed a well-developed and nourished woman. The heart was not enlarged; there were no murmurs. The lungs were clear and resonant; there were no rales. The blood pressure was 104 systolic, 60 diastolic, where it remained until July 1, when it was 134 systolic, 70 diastolic. On July 6 the urine showed a large trace of albumin and the blood pressure was 180 systolic, 100 diastolic; there was a great deal of edema. She was sent to the hospital, kept in bed and placed on a restricted fluid intake. Prior to delivery, the signs of toxemia cleared up, the blood pressure dropping to 130 systolic, 90 diastolic, and the urine containing only a very small amount of albumin.

On the morning of July 19, two days after delivery, the temperature was 99.2°F., and the pulse 84; the afternoon temperature was 100.6, the pulse 100; the uterus was larger than normal but not tender. On July 20 the temperature was 99.6°F. in the morning and 100.4 in the afternoon. On July 21 the temperature was 99.2°F. in the morning and 100.2 in the afternoon. On July 22, five days after delivery, the patient had a chill, following which the temperature rose to 102.0°F., and the pulse to 96. In view of the fact that the uterus was still large, the cervix was dilated, with the escape of a considerable amount of foul-smelling, thick, dark-brown lochia, and the uterus was curetted and washed out with a small amount of warm salt solution. Following this maneuver, the temperature rose to 103.0°F., and the pulse to 120. The following day, July 23, the temperature was 100.2°F. in the morning and 101.0 in the afternoon. On July 25, two and a half days after the uterus had been curetted, the patient had a chill lasting ten minutes; the temperature rose to 104.0°F., and the pulse to 140. The following morning the temperature had dropped to 99.2°F., and the pulse to 90, but another chill sent the temperature to 104.0 and the pulse to 140. On July 27 she had a chill in the afternoon, with a temperature of 104.0°F. and a pulse of 120.

The temperature continued to be of the picket-fence type for about ten days, when it came down to normal and remained there. She was discharged on August 16, one month after delivery.

*Comment.* It is impossible to be sure that intrauterine manipulation following a temperature for several days was the direct cause of precipitating a minor infection into a serious one; nevertheless such was probably the case. The patient was much sicker after the uterus had been curetted and washed out than she had been before. No cultures of the uterus were taken, nor were any blood cultures; no blood studies were made. The handling of this case was typical of that in vogue twenty-five years ago. The presence of the persistent low-grade temperature immediately after delivery until the chill on the fifth day is definite evidence of uterine infection starting soon after delivery. Today uterine cultures and blood cultures would have been taken, ice would have been applied to the fundus, some form of ergot would have been given to stimulate uterine drainage, but the uterus would never have been invaded. However, the patient was treated entirely conservatively after the initial mistake. It is quite fair to infer that if the uterus had been left alone, convalescence would have been much shorter; uterine trauma undoubtedly spread the infection.

This case also illustrates the successful treatment of toxemia occurring rather precipitantly late in pregnancy. Rest in bed and a restricted fluid intake were the essence of the treatment.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

## REPORT OF THE COMMITTEE ON INDUSTRIAL HEALTH

### HEALTH SERVICE IN INDUSTRY

An industrial medical service in order to justify its existence must accomplish the following objectives:

(1) It must by means of physical examinations assist

the employment department in the placement of applicants and in the weeding out of the very few who may be unfit for employment.

(2) It must co-operate with the safety engineer in making safe the plant and all its machinery and processes so that they may not constitute a source of injury or disease to any employee.

(3) It must furnish early and efficient care of industrial injuries, having in mind the welfare of the employee, the easing of his pain and the bringing about of his early return to work and full earning capacity.

(4) It must make the employee's working hours safe by giving him good healthful surroundings, good medical advice and protection from others who may have defects or disease which would constitute a menace to him.

(5) It should provide, so far as it is within the province of industry, reasonable protection from loss as a result of sickness or injury.

(6) It must co-operate with local public-health agencies, physicians and hospitals in carrying out the general community health program.

(7) It should maintain at all times strictly ethical and friendly relations with the family physicians of employees.

(8) It must be so conducted that the industrial physician always maintains with the employee the strictly confidential relation which traditionally exists between doctor and patient.

Health service in industry did not have its beginning in as comprehensive a program as this but even the least pretentious department today is attempting to live up to these requirements.

Facilities for the care of injuries today in our industrial plants are not considered adequate unless they are immediately available. A first-aid room with a nurse in charge may be adequate in some of the small plants, but all cases requiring more than first aid should be cared for by a physician. The physician may be the family doctor or a physician employed by the plant. In the large factories the great number of daily treatments necessary call for the employment of a full-time doctor.

The first step in this program is concerned with the choice of healthy employees by means of physical examinations at the time of employment. This examination should accomplish the following ends: first, place the man on the job for which he is physically fitted; second, protect the person already employed from the entrance into the plant of others who might have transmittable disease or who by some serious physical defect might constitute a menace if given employment; third, the discovery of remediable defects and the giving of helpful advice aimed toward their correction.

The second requirement in the establishment of a well-rounded program, according to the standards enumerated above, has to do with the bringing about of safe working conditions. This is a work which can be carried out in any plant whether or not that plant has a medical department. The trained safety engineer has by his excellent work in so many of our industries built himself a lasting monument in the reduction of accidents and the saving of lives. It is probably no exaggeration to say that most operations have now been made so safe that the majority of accidents now occurring are due to the faulty human element involved. The injured workman or some careless fellow-workman is frequently the cause of the accident. This weakness must not be overlooked. The employees must be educated in safe practices in all their activities, and this educational process is a logical function of the medical department as well as of the safety department. Recounting of the many ways in which these safety measures have been consummated is not necessary now but some of the devices are worthy of mention. These are: automatic brakes on revolving machinery, guards over gears and moving parts, hand-guards to prevent hands and fingers from entering the danger zone, hard toes for shoes to prevent injury from falling objects, automatic gates on elevators, exhaust ventilation for the removal of dust and fumes, establishment of safety zones by the efficient layout of traffic lanes, and many others just as important.

Frequent visits of inspection to all departments of the factory are essential to a successful health program of this type. The alert doctor can observe any infraction of the rules of safety and health and also at first hand see that his safety recommendations have been carried out.

Dust which contains silica in considerable quantity may constitute a hazard, and constant supervision of handling methods is necessary. Adequate exhaust ventilation and the use of respirators will as a rule prevent silicosis in the dusty trades, but only if someone makes it his job to see that these protective devices are used. There may be other hazards which cannot be guarded against by the methods above mentioned. Toxic materials and harmful dusts may constitute a definite hazard in some plants. It is the duty of physicians in these factories to know what materials are used and to determine their toxicity. It may in some instances be possible to substitute materials that are not toxic. When this is not possible, the physician must devise methods of protection. He should by careful, periodic examination of individuals exposed discover the earliest signs of harmful effect from any of these substances. Lead and benzol are materials of this type. There are of course many others.

The third requirement in this health program provides for the immediate care of the injured employee and can best be carried out by having a hospital or dispensary centrally located in the factory and made easily accessible to all employees. This should be so equipped as to be capable of caring for the most severe injury so far as first aid is concerned, and also for the continued care of all ambulatory cases. It is assumed of course that these treatments are made under adequate medical supervision. This medical supervision may be by a local practicing physician spending part time in the plant or by a physician devoting full time to the work.

The industrial physician will keep up to date on all the latest developments of medicine which pertain to his plant. Also the wise industrial physician will provide himself with a goodly number of consultants and hospitals in whose hands he can place those cases which he is not equipped to handle. In choosing these consultants and hospitals he will invariably pick the best, and as a consequence the industrial employee is provided with the finest medical service obtainable. This type of service should be provided by industry, not from the standpoint of good economics alone, but from a real interest in the welfare of the workers. Most industrial workers appreciate this fact and have come to look upon such service as a matter of right. They enjoy these benefits and are appreciative of them.

The medical and surgical care of these injuries calls for a thorough knowledge of the plant set-up on the part of the physician who is responsible for this service. He must know how the injury occurred in order to assist the safety department to guard against its recurrence.

He must know the physical requirements of each injured employee's job so that he may return him to productive work as soon as he is able to do so. Much economic loss may be prevented if he does this part of his job well.

The fifth step of the program has to do with protection from financial loss due to sickness and injury. It is not very far back to the time when the worker was not compensated for his injuries unless they were due to neglect on the part of the employer, and then it was often necessary for him to sue at common law in order to obtain compensation. The advent of the Workmen's Compensation Law and its acceptance by most industries have eliminated that trouble, and now employees are usually compensated without litigation, regardless of who is to blame for the injury.

There is much more time lost from work because of non-industrial sickness and non-industrial accident than as a result of industrial accidents. This also calls for some type of protection for the employee against financial loss. Mutual-benefit plans, non-contributory benefit plans entirely financed by the employer and contributory group-insurance plans are those commonly resorted to at present. Mutual-benefit plans are functioning well in some plants and may be the plan of choice for a particular group. This plan has the advantage of giving the employees a direct interest in the management of it. A contributory plan, such as is furnished by group-health and accident insurance, fulfills the requirements and is not apt to be discontinued because of lack of funds to carry it on. In this type of insurance the employer can still share part of the cost. A popular group-insurance plan today provides weekly indemnity in the event of sickness or non-industrial accident, usually for a maximum period of thirteen weeks for any one disability. There is a waiting period of one week before benefits begin, so that no disability short of eight days receives payment. A life-insurance policy may be issued in combination with the weekly indemnity. This type is usually underwritten by an insurance company but in many cases the administration of it is a function of the medical department.

Any of these plans call for investigation of absenteeism by the medical department. Probably the best investigator of absentees is a trained nurse who is usually well known to all employees and is well received when she visits their homes. Being well trained to recognize illness and need, she is able to help in obtaining proper medical assistance for the patient by co-operating with the family physician or other interested agencies. The majority of employees accept this investigation at its true value and desire its continuance.

There can be no doubt in the minds of anyone as to the value of such insurance. Those who are close to the factory worker and his financial problems know what a staggering blow is dealt by an extended absence from work, especially when the disabled one is the head of a family. Not only has he the expense of medical care but he must find in addition a way to provide his family with the necessities of life while he is cut off from his earnings. Provision must be made to reimburse, in part at least, the disabled employee. The question is often asked whether or not employees take advantage of a plan like this to take vacations with pay. It is true that an occasional one may malinger, but there is nothing gained by penalizing the majority for the misdemeanors of the few. All plans have flaws and if the opportunity to malinger is the flaw in this type of insurance it still fulfills its purpose. It should be continued until something better offers.

The sixth requirement of the medical department in industry is co-operation with existing outside agencies, family physicians, boards of health and welfare organizations. Though the physician is doing much for the worker through industrial health programs he has not replaced these other agencies. He has merely augmented their services and is not self-sufficient. The family doctor is just as necessary to employees to whom this service is available as he was before. He is even more necessary, because industrial medical service has educated employees to take better care of their health and has encouraged them to seek the aid of their family doctors for conditions which they had been accustomed to ignore.

The industrial physician has an opportunity to do a real service to the community in his work along the lines of preventive medical care. The early discovery of communicable disease and the use of all known methods for its control are two of the principal functions of an industrial medical department. In protecting employees from exposure to them he is also protecting the community. The next great step in the fight against tuberculosis may possibly be made through the co-operation of industry and the departments of public health. Great work has been done in the young age groups in the public schools but less among employed groups by public-health agencies. The doctors in industry can be of help by adopting in the factories the same methods which have accomplished so much among the young groups. Through their industrial clinics they should be able to assist greatly in the continuation of this program.

This in general is the aim of most industrial medical departments, some giving service beyond that set forth here and some giving much less. The industrial physicians who are responsible for this service, however, must be well trained and especially familiar with occupational hazards and the general practice of safety and must not lose sight of the fact that no matter how large their organizations may be the confidential relation of physician and patient must be maintained.

W. Irving Clark, M.D., *Chairman*,
Louis R. Daniels, M.D.,
Noel G. Monroe, M.D.,
Halstead Murray, M.D.

## DEATHS

HILLARD — James P. Hillard, M.D., of Springfield, died January 29. He was in his sixty-third year.

Born in Fall River, he received his degree from Jefferson Medical College of Philadelphia in 1903, and had practiced in Springfield for thirty-seven years. Dr. Hillard was examiner for the Bureau of Pensions, Department of the Interior, and for the Travelers Insurance Company. For thirty-three years he was medical inspector in the public schools of Springfield and had served as physician for the Police and Fire departments.

He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow, a brother, a niece and a nephew survive him.

KITTREDGE — Joseph Kittredge, M.D., of North Andover, died January 31. He was in his eighty-third year.

Dr. Kittredge received his degree from the Harvard Medical School in 1880 and practiced medicine in Brookline and Marblehead until 1918 when he retired to run his own sanatorium. He was the eighth in a nine-generation family of doctors.

He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow, a brother, two sons and a daughter, Dr. Edwina Kittredge Newberry, survive him.

PAYNE — JAMES H. PAYNE, M.D., of Allston, died January 31. He was in his seventy-eighth year.

Born in Boston, he attended Harvard University and received his degree from Harvard Medical School in 1889. He studied in London and Paris, and practiced medicine in Boston for eight years before entering the naval corps.

Dr. Payne was a fellow of the Massachusetts Medical Society, and the American Medical Association and held memberships in the Association of Military Surgeons of the United States and the British Medical Association.

His widow, a son and a daughter survive him.

PRATT — DAVID D. PRATT, M.D., of New Bedford, died January 29. He was in his sixty-first year.

Born in Wisconsin, he attended Dartmouth College and received his degree from the Harvard Medical School in 1906.

Dr. Pratt was a fellow of the Massachusetts Medical Society, the American Medical Association and the American College of Surgeons. He was also a member of the New Bedford Medical Society.

His daughter, a son, a brother and a niece survive him.

WHITESIDE — GEORGE S. WHITESIDE, M.D., of New York City, died January 29. He was in his sixty-eighth year.

Born in Boston, he attended Harvard University and received his degree from Harvard Medical School in 1897. He was formerly examining physician for several insurance companies and had served as surgeon for the Winchester Arms Company of New Haven.

Dr. Whiteside was a former member of the Massachusetts Medical Society and was a fellow of the American Medical Association and the American College of Surgeons. Formerly he was secretary of the Harvard Medical Alumni Association.

His widow, two sons and a daughter survive him.

## CORRESPONDENCE

### PATHOLOGICAL SERVICE FOR THE SMALL HOSPITAL

*To the Editor:* Recently a group of small hospitals near Boston requested the New England Pathological Society to aid them in obtaining the services of a pathologist. This was the first time in its history that the society had been directly approached by a hospital for such a purpose, and it was believed that perhaps this was the beginning of an opportunity for the society to act as an impartial adviser to hospitals with respect to their pathological services, only of course when directly solicited. The society has therefore appointed a committee, the members of which will act as consultants and as a clearing house to help both hospitals and pathologists.

Any request for information should be forwarded to the secretary.

BENJAMIN CASTLEMAN, *Secretary,*
New England Pathological Society.

Massachusetts General Hospital,
Boston.

### A SURE CURE

*To the Editor:* Below is a copy of an item taken from Middlebrook's *New England Almanac* for the year 1880 which I thought might be of interest to readers of the *Journal.*

> An exchange says: Every little while we read in the papers of someone who has stuck a rusty nail in his foot, or knee, or hand, or some other portion of his body, and that lockjaw resulted therefrom, of which the patient died. If every person was aware of a perfect remedy for all such wounds, and would apply it, then all such reports must cease. Yet all these wounds can be healed. The remedy is simple, almost always on hand, and can be applied by anyone, and, what is better, it is infallible. It is simply to smoke the wound that is inflamed with burning wool or woolen cloth. Twenty minutes in the smoke of wool will take the pain out of the worst wound, repeated two or three times, it will allay the worst cases of inflammation arising from a wound.

CHARLES W. ROBERTSON, M.D.

Amherst, Mass.

### RESTORATION OF LICENSE

*To the Editor:* This is to inform you that in accordance with the vote of the Board of Registration in Medicine, the license of Dr. Rafael R. Garcia, of Agawam, revoked on September 29, 1938, was restored on January 18, 1940.

STEPHEN RUSHMORE, M.D., *Secretary.*

State House,
Boston.

## NOTICES

### ANNOUNCEMENTS

JACOB KAMINSKY, M.D., announces the opening of an office at 891 Massachusetts Avenue, Cambridge.

HYMAN H. SHUMAN, M.D., announces the opening of an office at 107 Prichard Street, Fitchburg.

### BOSTON CITY HOSPITAL

The monthly clinicopathological conference will be held at the Boston City Hospital on Wednesday, February 14, at 12 o'clock noon, in the Pathological Amphitheater.

### PETER BENT BRIGHAM HOSPITAL

A joint medical and surgical clinic at the Peter Bent Brigham Hospital will be held on Wednesday, February 14, from 2 to 4 p.m. Drs. Elliott C. Cutler and Soma Weiss will speak on "Nervousness."

### BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

The next meeting of the Boston Society of Psychiatry and Neurology will be held at the Boston Medical Library on Thursday evening, February 15, at 8:15.

PROGRAM

A Phantasy Gratified by a Suicidal Act. Dr. Ives Hendrick.

Prefrontal Lobotomy in Two Patients with Agitated Depression: With therapeutic evaluation and further reference to prefrontal-lobe function. Drs. W. J. Mixter, K. J. Tillotson and David Wies.

## HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held on Tuesday, February 13, in the amphitheater of the Peter Bent Brigham Hospital (Shattuck Street entrance), at 8:15 p.m.

PROGRAM

Presentation of cases.

The Application of Medical Science to the Administration of Justice. Dr. Alan R. Moritz.

Medical students and physicians are cordially invited to attend.

## NU SIGMA NU LECTURE

The annual lecture sponsored by the Harvard chapter of Nu Sigma Nu will be given in Building E of the Harvard Medical School on Thursday, February 29, at 5:00 p.m. Dr. Donald D. Van Slyke, of the Rockefeller Institute, will speak, his subject being "Renal Physiology."

## LAWRENCE CANCER CLINIC

The regular Lawrence Cancer Clinic, to be held at Lawrence General Hospital, 1 Garden Street, Lawrence, on Tuesday, February 20, at 10:00 a.m., will be a demonstration and teaching clinic for physicians, with Channing C. Simmons, M.D., of Boston, as consultant. Physicians of the north half of Essex County are invited to accompany any of their patients whom they desire to have this service or to send them with a note. A report will be returned to every physician who sends a patient. The service is gratis. Any physician is welcome to attend the clinic.

## SUFFOLK CENSORS' MEETING

The censors of the Suffolk District Medical Society will meet for the examination of candidates at the Boston Medical Library, 8 Fenway, Boston, on Thursday, May 2, at 4:00 p.m.

Candidates should make personal application to the secretary and present their medical diplomas at least six weeks before examination (if graduates of foreign medical schools or schools not on the list recognized by the Council, at least eight weeks before).

## WORCESTER DISTRICT MEDICAL SOCIETY

The next meeting of the Worcester District Medical Society will be held at the Worcester State Hospital, Worcester, on Wednesday, February 14. Dinner will be served by the hospital at 6:30 p.m. Following dinner there will be a business and scientific meeting. Dr. William Malamud will present a paper on "The Treatment of the Neuroses in General Practice." Saul Rosenzweig will speak on "Experimental Neuroses," and the talk will be illustrated by Maier and Glazer's movie film which was awarded a prize by the American Association for the Advancement of Science.

## NEW ENGLAND PATHOLOGICAL SOCIETY

The next meeting of the New England Pathological Society will be held on Thursday evening, February 15, at 8:00 at the Tufts College Medical School, 416 Huntington Avenue, Boston.

PROGRAM

Pathological Observations in Simmonds' Disease. Dr. R. C. Wadsworth.

Immediate Wheal and Erythema Type Reactions to Simple Chemical Substances. Dr. John Jacobs.

Pathological Findings in Leukoerythroblastic Anemia. Dr. S. B. Thorson.

Primary Tumor of the Heart. Dr. Harold Wood.

The pathological demonstrations will be available at 7:00 p.m. There will be a short business meeting followed by a collation.

Physicians and students are cordially invited to attend.

## UNITED STATES MARINE HOSPITAL

The staff meeting of the United States Marine Hospital, Chelsea, will be held at "The Hut," on Friday afternoon, February 16, at 4:00. Dr. John W. Strieder will speak, his subject being "Clinical Aspects of Bronchogenic Carcinoma."

## HATHERLY MEDICAL CLUB

There will be a meeting of the Hatherly Medical Club at the Toll House on Wednesday afternoon, February 14, at 3:00. Dr. William E. Browne will be the guest speaker, his subject being "Diagnosis and Treatment of Nerve and Tendon Injuries to the Hand."

## VAN METER PRIZE AWARD

The American Association for the Study of Goiter again offers the Van Meter Prize Award of three hundred dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The award will be made at the annual meeting of the association which will be held at Rochester, Minnesota, on April 15, 16 and 17, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or research investigations, should not exceed three thousand words in length and must be presented in English; a typewritten double-spaced copy should be sent to the corresponding secretary, Dr. W. Blair Mosser, 133 Biddle Street, Kane, Pennsylvania, not later than March 15.

A place will be reserved on the program of the annual meeting for presentation of the prize essay by the author, if it is possible for him to attend. The essay will be published in the *Proceedings* of the Association. This will not prevent its further publication, however, in any journal selected by the author.

## ALUMNI DAY, NEW YORK UNIVERSITY COLLEGE OF MEDICINE

Alumni Day of New York University College of Medicine will be held on February 22. Following opening remarks by Dr. James S. Smith, president of the Alumni Association, a series of formal lectures on "Modern Aspects of Preventive Medicine" will be presented at the medical school during the morning and afternoon sessions. Luncheon will be served at the school at 1:00 p.m. Dean McEwen will be host at an informal reception in the dean's office at 5:00 p.m.

## SOCIETY MEETINGS AND CONFERENCES

### Calendar of Boston District for the Week Beginning Sunday, February 11

Sunday, February 11

4 p.m. Cancer. Dr. Grantley W. Taylor. Free public lecture. Harvard Medical School, amphitheater of Building D.

4 p.m. Cancer of the Stomach. Dr. Marshall K. Bartlett. Illustrated, public, health lecture. Faulkner Hospital auditorium.

Tuesday, February 13

*9–10 a.m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

8:15 p.m. The Obstetric Pelvis. Dr. W. E. Caldwell. Journal Club. Boston Lying-in Hospital.

*8:15 p.m. The Application of Medical Science to the Administration of Justice. Dr. Alan R. Moritz. Harvard Medical Society. Peter Bent Brigham Hospital amphitheater (Shattuck Street entrance).

Wednesday, February 14

*9–10 a.m. Clinicopathological conference. Dr. C. S. Keefer. Joseph H. Pratt Diagnostic Hospital.

12 m. Boston City Hospital, monthly clinicopathological conference.

*2–4 p.m. Nervousness. Drs. Elliott C. Cutler and Soma Weiss. Peter Bent Brigham Hospital.

Thursday, February 15

*8 p.m. New England Pathological Society. Tufts College Medical School.

8:15 p.m. Boston Society of Psychiatry and Neurology. Boston Medical Library.

Friday, February 16

*9–10 a.m. Some Observations on Pituitary Adenoma. Dr. M. C. Sosman. Joseph H. Pratt Diagnostic Hospital.

Saturday, February 17

*9–10 a.m. Hospital case presentation. Dr. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*Open to the medical profession.

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February 9 — William Harvey Society. Page 203, issue of February 1.

February 11 — Public lecture. Salem Hospital. Page 1042, issue of December 28.

February 11 — Free public lecture. Quincy City Hospital. Page 77, issue of January 11.

February 11–14 — International College of Surgeons. Page 759, issue of November 9.

February 14 — New England Dermatological Society. Page 203, issue of February 1.

February 14 — Hatherly Medical Club. Page 244.

February 16 — Staff meeting. United States Marine Hospital. Page 244.

February 20 — Lawrence Cancer Clinic. Page 244.

February 20 — South End Medical Club. Page 202, issue of February 1.

February 22 — Alumni Day. New York University College of Medicine. Page 244.

February 22–24 — American Orthopsychiatric Association. Page 957, issue of December 14.

February 29 — Nu Sigma Nu Lecture. Page 244.

March 2, June 8 and 10 — American Board of Ophthalmology. Page 719, issue of November 2.

March 7–9 — New England Hospital Association. Hotel Statler, Boston.

March 14 — Pentucket Association of Physicians. 8:30 p.m., Hotel Bartlett, Haverhill.

April 15–17 — American Association for the Study of Goiter. Page 203, issue of February 1.

April 24–26 — Scientific Session. Academy of Physical Medicine. Hotel John Marshall, Richmond, Virginia.

May 10–18 — American Scientific Congress. Page 1043, issue of December 28.

May 13 — United States Pharmacopoeial Convention. Page 202, issue of February 1.

June 7–9 — American Board of Obstetrics and Gynecology. Page 1019, issue of June 15.

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### District Medical Societies

ESSEX SOUTH

February 14 — Cough, Sputum, Hemoptysis — How shall they be investigated? Dr. Reeve H. Betts. Essex Sanatorium, Middleton.

March 6 — Experimental and Clinical Considerations of Sulfanilamide Treatment of Hemolytic Streptococcus Infections. Dr. Champ Lyons. Lynn Hospital, Lynn.

April 3 — Addison Gilbert Hospital, Gloucester.

May 8 — Annual meeting. Salem Country Club, Peabody.

FRANKLIN

March 12 — Franklin County Hospital, Greenfield.

May 14 — Franklin County Hospital, Greenfield.

HAMPSHIRE

March 13.

May 8.

Meetings are held at 11:30 a.m. at the Cooley Dickinson Hospital, Northampton.

MIDDLESEX EAST

March 20.

May 15.

Meetings are held at 12:15 p.m. at the Unicorn Country Club, Stoneham.

MIDDLESEX NORTH

April 24.

July 31.

October 30.

NORFOLK SOUTH

March 7.

April 4.

May 2.

All meetings, with the exception of one which is usually held at the Quincy City Hospital, are held at the Norfolk County Hospital in South Braintree, at 12 o'clock noon.

PLYMOUTH

March 21 — Goddard Hospital, Brockton.

April 18 — State Farm.

May 16 — Lakeville Sanatorium, Lakeville.

SUFFOLK

March 27 — Scientific meeting. Symposium on Ulcerative Colitis and Diarrheas. Under the direction of Dr. Chester M. Jones.

April 24 — Annual meeting in conjunction with the Boston Medical Library. Election of officers. Program and speakers to be announced later.

May 2 — Censors' meeting. Page 244.

WORCESTER

February 14 — Page 244.

March 13 — Worcester Memorial Hospital.

April 10 — Worcester Hahnemann Hospital.

May 8 — Worcester Country Club.

Each meeting begins with a dinner at 6:30 p.m. and is followed by a business and scientific meeting.

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## BOOKS RECEIVED FOR REVIEW

*Clinique et physiopathologie des maladies coeliaques.* Robert Dubois. 349 pp. Paris: Masson et Cie, 1939. 80 Fr. fr.

*Cardiovascular-Renal Disease: A clinicopathologic correlation study emphasizing the importance of ophthalmoscopy.* Lawrence W. Smith, Edward Weiss, Walter I. Lillie, Frank W. Konzelmann and Edwin S. Gault. 227 pp. New York and London: D. Appleton-Century Co., 1940. $4.50.

*Lane Medical Lectures: Viruses and virus diseases.* Thomas M. Rivers. 133 pp. Stanford University, California: Stanford University Press, 1939. $2.50.

*An Introduction to Gastro-Enterology.* Walter C. Alvarez. Being the third edition of *The Mechanics of the Digestive Tract.* 778 pp. New York and London: Paul B. Hoeber, Inc., 1940. $10.00.

*Citrus Fruits and Health: Clinical and scientific data from the literature of nutrition as related to citrus fruits.* 73 pp. Lakeland, Florida: Florida Citrus Commission, 1939. Complimentary.

*Fundamentals of Biochemistry: In relation to human physiology.* T. R. Parsons. Sixth edition. 461 pp. Baltimore: William Wood & Co., 1939. $3.00.

*Surgical Diagnosis.* Stephen Power. 228 pp. Baltimore: William Wood & Co., 1939. $4.50.

*Massage and Remedial Exercises: In medical and surgical conditions.* Noël M. Tidy. Fourth edition. 458 pp. Baltimore: William Wood & Co., 1939. $5.25.

*Demonstrations of Physical Signs in Clinical Surgery.* Hamilton Bailey. Seventh edition. 310 pp. Baltimore: William Wood & Co., 1940. $6.50.

*Standard Methods of the Division of Laboratories and Research of the New York State Department of Health.* Second edition. 681 pp. Baltimore: Williams & Wilkins Co., 1939. $7.50.

## BOOK REVIEWS

*Psychopathic States.* D. K. Henderson. 178 pp. New York: W. W. Norton & Co., Inc., 1939. $2.00.

The problem of that heterogeneous and ill-defined psychiatric group known as the constitutional psychopathic inferior is of importance, inasmuch as the conduct of the members of this group is often such as to bring them into conflict with the law and consequently involves psychiatric expert testimony, and it is this problem that Dr. Henderson, the eminent British psychiatrist, considers. His discussion has charm and wisdom, and he comes to the predestined conclusions that much more research and more exact studies must be done before the conditions described under this widespreading term can be understood and socially or psychiatrically treated.

The constitutional psychopathic inferiors constitute a group in which the conduct disorder and general psychological reactions run the gamut from the liar-swindler type to the less numerous and more bizarre individuals in whose life homosexuality, rape and ferocious immorality appear, and whose responsibility consequently must be passed on. In between these extremes of conduct, one meets the chronic alcoholic, the drug addict, the ne'er-do-well and those unemployable individuals whose difficulty is not due to physical disease but to a mental state which cannot be classified as psychotic or neurotic but comes down to instability of personality of one type or another.

It will be seen that there can be no sharp differentiation between the normal and these individuals. Homosexuality and other abnormal sexual manifestations are widespread and involve individuals whose lives otherwise run not only along normal lines but sometimes along supernormal ones. Similarly, alcoholism cannot be linked with social or psychological inferiority since among the alcoholics one often finds the geniuses of the race.

Indeed, Dr. Henderson brings up the question which for so long a time has been a focus of human attention, — whether or not genius is a psychopathic trait, — to which Lombroso, Nordau and Henderson, as well as many others, have answered "Yes," and other workers, especially the eugenists, "No." As a matter of fact, generalizations can no more be made about the trait of genius than about the qualities of the so-called "normal" individual, since there are many kinds of genius, and high ability of whatever type may be linked to all kinds of qualities of character and personality. The personality of man is not a harmonious composition, no more than his body is. Character is largely a mosaic in which there is often a juxtaposition of the most incongruous qualities. A Bacon may be a great philosopher and a wise lord chancellor, but he may also be a bribe-taker and a foolish thief.

Henderson definitely indicates all this. The point of view he expresses is pragmatic and clinical, but he has as yet no solution, either legally or otherwise, for the problems inherent in the term, constitutional psychopathic inferiority, and in the types of individuals classed as suffering from it. However, he makes the important observation that the law in England has commenced to modify its attitude toward this group; that the concept of limited responsibility is appearing in the court decisions; and that the all-or-none theory of responsibility is passing away, as indeed it should, since there are degrees and shades of responsibility for crime as there are degrees and shades of all human qualities and attributes.

A deeper problem has preoccupied the reviewer for many years. It is assumed that adaptation to whatever happens is the test of normality, and in a certain sense this is true. But human society has built up extraordinary social mechanisms to which the individual must adapt himself; furthermore, it has built up systems of reward for certain types of personality and punishment for others. There is nothing in any candid view of society which indicates that it has shown much wisdom in the selection of either the types it rewards or the types it punishes. Thus, the acquisitive type of human being is over-rewarded. The individual who sacrifices biological necessities, such as reproduction, often has a social advantage over the individual who reproduces freely, as from a biological point of view he should. The person who sacrifices certain social trends in his nature, such as friendliness and generosity, often succeeds, while the individual who expresses these qualities in his life may become a social failure, and so forth. There may be a psychopathic society to which normal individuals cannot adapt themselves and to which certain abnormal individuals are able to adapt themselves. It is not proved that either social success or failure is necessarily a criterion of essential normality or abnormality, or indeed of personal worth or worthlessness.

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*Midwifery.* By ten teachers, under the direction of Clifford White. Edited by Comyns Berkeley, Clifford White and Frank Cook. Sixth edition. 676 pp. Baltimore: William Wood & Co., 1938. $6.00.

This sixth edition is a textbook on obstetrics by ten English obstetricians. The book is written primarily for English students preparing for their final examinations. It has little value from the American standpoint except for the purpose of comparing English methods with those practiced in America. As a reference book it is of value.

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*Tests for Mental Development: A complete scale for individual examination.* F. Kuhlmann. 314 pp. Minneapolis, Nashville, Philadelphia: Educational Publishers, Inc., 1939. $2.00.

The author of this book is a well-known authority on the subject of mental tests. He presents, for the specialist in the field of psychology, a new scale of tests, based on the Heinis mental-growth curve. Practically none of the original Binet tests are used. Some from the author's previous revisions of the Binet scale of 1912 and 1922 and from the Kuhlmann-Anderson group tests are included in modified form. The rest are derived from other sources. The new scale seems to remove some of the disadvantages of the Binet type of scale. The application of these tests in the way that they are intended to be used, however, requires a long period of training and familiarity with the general scope of mental tests.

Workers in the field of psychology will find some of the author's suggestions to be of interest, — for example, his recommendation that the "personal constant" of Heinis be used in place of the I.Q. as a measure of intelligence, — but it is doubtful that the new scale will be of value in the hands of the medical man.